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THE INHERITANCE OF MENTAL DISEASES

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BY

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To

WILLIAM WASHINGTON GRAVES

teacher and friend, to whose enthusiasm and insight in the study of families the author owes his own interest in the subject

THE INHERITANCE OF MENTAL DISEASES

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PART I

INTRODUCTION

That we are not yet arrived at a resting place in our knowledge of mental diseases is a fact no student of the subject matter of psychiatry can deny. We understand a few mental diseases quite well but a large number of the cases that come before the psychiatrist either in private practice or in the hospital are fundamentally unknown to his science, unknown at least from the standpoint of cause and of treatment. In a rough way he can predict the course of the disease process, whether towards recovery or towards deterioration, but even here he has no absolute criteria for his judgment and it is never a surprise to him when events prove him wrong.

So it may seem rather hazardous to write a book on the transmission of mental diseases, and it is hazardous. But though one may be wrong in every conclusion reached and even in every premise there is a justification for marshalling the facts of the situation as seen after years of investigation and honest effort; there exists a right for a humble devotee of science to criticize, perhaps too frankly, other men's opinions and attitudes, to present his own views and to point the way for better work. On this basis this book is written, and I claim no more or less for it than that it is a faithful record of labor and thought, and that it hopes to be a guide and a spur to those equipping themselves for a similar task.

Because I wish to claim the attention of others than the professional psychiatrist, though his opinion is mainly in my mind, I shall attempt to outline the situation in psychiatry so as to make it intelligible not only to the physician in general practice or engaged in non-psychiatric work, but also to the biologist, the social worker and the zealous layman of whatever work who finds the transmission of mental diseases a subject sufficiently important to claim his time and attention. Since the transmission of mental diseases, commonly called the inheritance of insanity, is one of the great subjects discussed by the eugenists and those interested in race betterment, it is important that the facts (or opinion) of any writer be made accessible. Indeed one need no longer apologize for popularizing his professional work, for the day is past when the medical scientist sits aloof in his consulting room or laboratory, watching the world go by.

This book will, then, deal with the following subjects:

First, it will demonstrate that the term insanity is a hindrance to the proper understanding of mental diseases, and that with the thrusting of this term into an outer limbo we shall thrust such notions as that mind and body are separate, and that mental diseases are anything else than diseases of the human being, to be studied in a naturalistic manner, and clinically. I shall adopt the belief that there are independent groups of mental diseases, which like animals of quite different biological nature happen to be in the same zoo under the same trainer. Some of these diseases will be declared to have no "hereditary" value, and others to "run in families" as the phrase goes.

Second, certain outstanding facts about mental diseases will be discussed; the marriage rate of various groups; the relation of sex to family mental disease; the way diseases go from generation to generation, and the path they take in the same generation. This part, long and perhaps tedious, will be taken mainly from my previous publications but reviewed by a riper experience.

Third, the various theories of the "heredity" of mental diseases will be considered, and criticized. Mainly I shall limit myself to the theory called "polymorphism," the Mendelian theory, and the less known idea of germplasm injury, called by Forel "blastophoria."

Fourth, this will bring up the question, are the transmissible mental diseases really hereditary characters, in the sense that stature and blue eyes are hereditary characters, or do they merely represent diseases, caused by unknown agents whose effects persist over two or more generations and from which a stock may either die or recover? I shall adopt the latter hypothesis and shall adduce a slender stock of evidence not so much in its support as for its possibility.

Fifth, and finally, we shall come to the larger problem that is involved by the issue of heredity or disease. For if a true heredity is involved then our humanitarian efforts are dysgenic, cacogenic, i.e., are injurious to the race. Hygiene, which has as its aim the preservation of the individual, is bad for the stock, so the eugenists believe and so indict the noblest efforts of our civilization. But suppose they are wrong, that the race is injured by adverse environmental conditions, and that such injury may persist for generations; then our *social* efforts are *racial* efforts and our noblest strivings have a eugenical backing. I shall endeavor to make a case for this belief though confessedly drawing in some part upon my hopes as well as upon facts.

CHAPTER I[1]

"An Idol of the Market Place," the Term Insanity

But the Idols of the Market place are the most troublesome of all; idols which have crept into the understanding through the alliances of words and names. For men believe that their reason govern words, but it is also true that words react on the understanding; and this it is that has rendered philosophy and the sciences sophistical and inactive. Now words, being commonly framed and applied according to the capacity of the vulgar, follow those lines of division which are most obvious to the vulgar understanding. And whenever an understanding of greater acuteness or a more diligent observation would alter those lines to suit divisions of nature, words stand in the way and resist the change. Whence it comes to pass that the high and formal discussions of learned men end oftentimes in disputes about words and names; . . . .

The idols imposed by words on the understanding are of two kinds. They are either names of things which do not exist (for as there are things left unnamed through lack of observation, so likewise are there names which result from fantastic suppositions and to which nothing in reality corresponds), or they are names of things which exist, but yet confused and ill-defined, and hastily and irregularly derived from the realities.

When Francis Bacon thus characterized the difficulties that confront our thought he assuredly had not in mind the term "Insanity." Yet everything he has said applies with extraordinary emphasis to this word, for it has hindered the progress of Psychiatry to a great degree. When a man is asked to define insanity, he can only say "the state of being insane" or else "unsound mentally" neither of which mean anything concrete. He may go on to speak of the symptoms of "insanity" such as delusions, hallucinations, emotional changes, etc., but finally he has to take refuge in the statement that there are many kinds of insanity, when he might better say that there are many mental diseases, having totally different causes and different courses, and the main relationship these diseases have one to the other is that *amongst* their symptoms are mental changes, which tradition and the law have labelled insanity.

[1] The preparation of this manuscript was financed by a grant of money from the Massachusetts Commission for Mental Diseases. My thanks are due to the director, Dr. George M. Kline, for his generosity in this matter.

Insanity is a term which has its roots in that period when men thought that the insane were possessed by devils, or when the moon was thought to cause mental changes, and it has the same kind of value that the words "lunacy" and "lunatics" have. At a later date the law stepped in to define insanity because property rights and the safety of the insane and the safety of others had to be conserved. Since the treatment of the insane is in part custodial and since such custody is essentially the deprivation of liberty and freedom in their most intimate and precious meaning, the law had to be invoked in this sphere of medicine as in no other. But the law is conservative, based on the conclusions and procedures of yore, is not interested in clinical distinctions and differences, and wants only to know "Does this man know the difference between right and wrong" (who does?), "does he know the distribution of his property or properties," "is he dangerous to himself or others?" The law is not interested in mental diseases, it cares not whether a man has dementia praecox or dementia paralytica and yet these two diseases are very far apart both in cause, pathology, treatment and hereditary importance. In other words though *biologically* and medically the mental diseases belong in separate categories, *legally and in the common mind* they are fused together in "insanity."

It is this relationship of law and common thought to the word insanity, and to any substitute word, which makes it useless and pernicious to psychiatry and to the discussion of heredity. For we have split up the fused mass of the insane, as the untrained mind and the past time saw it, into diseases of varying cause, pathology and course. To use the term insanity as if there were an entity corresponding to it is as if we thought all diseases of respiration were the same, and had a term, let us say, dyspnoea, for the general state of lung trouble. But we know that penumonia, bronchitis, asthma, pulmonary tuberculosis, lung abscess, and hypostatic pneumonia are separate entities, having little or no relationship to one another, save as they have some symptoms in common. We have made progress in medicine not so much through *unifying* diseases and disease phenomena as by *isolating* them, by discovering that syphilis and gonorrhoea are *not* the same disease, though the great John Hunter declared they were;[2] by discarding the term scrofula

[2] Indeed the term Insanity has the same sort of value as the term venereal disease. Gonorrhea and syphilis are totally different diseases though both

and demonstrating that many diseases from tuberculosis to anaemia were fused under the term; by isolating the enteric fever of our ancestors into typhoid and paratyphoid; by laboratory specific-reactions for various diseases as the Wassermann reaction for syphilis, the Pirquet test for tuberculosis, the Widal test for typhoid and the Schick reaction for diphtheria.

Moreover the idea of insanity[3] is not at all parallel to the idea of mental diseases. A man may have general paresis, which is one of the most common of the mental disease, and yet not be "insane." By indisputable medical criteria he may be a victim of this disease, yet legally he may not be at all sick, since he may have no noticeable defect of judgment or disorder of conduct. Similarly with dementia praecox, manic depressive insanity and paranoia—there may be years of the mental disease but no insanity in that the patient can pass muster as a sane man by the legal standards. Though in the case of the above mentioned diseases the majority of patients finally reach the status of insanity there are other mental diseases where the great majority of patients and their physicians would fiercely resent the idea of mental disease at all and where insanity is only occasionally declared to exist. These diseases are known medically as the functional neuroses; neurasthenia, psychasthenia, and hysteria, and yet nothing is so purely mental as the fixed ideas, the fears, the paralyses, impulsions, and anaesthesiae of these conditions. Because we associate the term mental diseases with insanity we are loathe to call things by their right names, and so fall into vicious mental habits.

The great Spencerian formula of evolution applies with remarkable pertinence to the evolution of our psychiatry. Formidably expressed the formula is in reality simple, and points out that an ill-defined homogenity changes gradually into well defined heterogenities. So in psychiatry—we have started with an ill-defined, obscure, mystical homogenity; insanity. As our subject has ap-

are most commonly acquired through sexual relationship. But there the resemblance ends—and there is more resemblance between tuberculosis and syphilis than there is between syphilis and gonorrhea. Further, though gonorrhea and syphilis are called venereal diseases this is but a superficial term. Gonorrhea may and does often involve joints and heart, while syphilis starting as the venereal disease involves every tissue of the body.

[3] For a fascinating discussion of the subject of insanity as here presented see Southard and Jarrett, "Kingdom of Evils" (182).

proached a science and we have thrown off the traditions which hampered, the ill-defined homogenity has become broken up into well-defined mental diseases. While it is well to seek for underlying unities it is wise to remember that progress, at least for a long time, will lie in the direction of splitting up "unities" into clinical units.

MIND AND BODY

If we drop the conception of insanity and insane and speak of mental diseases we find that there are many distinct groups of diseases having biologically less relationship to one another, except for two groups, than to certain diseases of the body. And we must discard also the idea that psychiatry deals with mind-disease, for there is no theory of separation of the organism into mind and body which has a medical-biological reason for existing. Mind is not even at all a brain function, it is a function of the entire organism and has its roots in the viscera, the endocrinal glands, the vegetative nervous system and even in the muscles and joints.[4] Mood is a matter as much of intestines as of brain, emotions are possibly more related to viscera and to endocrines than to cerebrum, and vigor, out of which springs hope, courage, clearness of thought and pertinacity of attention is a matter of fatigue, health, age, arteries, etc. Desire has one of its origins in alteration of the body tissue and there are innumerable instances of character change, personality alteration, mind metamorphoses that are clearly somatic in their causation. Some of these will be referred to as we go further, but for details of this the reader must go to other volumes. To quote Conklin (31).

> Indeed the entire organism structure and function, body and mind, is a unity, and the only justification for dealing with these constituents of the organism as if they were separate entities, whether they be regarded in their adult condition or in the course of their development is to be found in the increased convenience and effectiveness of such treatment.

So when we use the term "*mental* diseases" we are using a convenient method of expression which denotes that the *striking symptoms* are mental changes, though there may be and often are as important changes in the organs and tissues of any part of the body, and the cause may be, and in some cases is, disease of non-nervous parts

[4] I refer the reader to Watson's (209) Behaviorist Psychology, White's (215) Foundations of Psychiatry, and Myerson's (142) Foundations of Personality.

whose well-being is essential to proper mental functioning. Thus we speak of myxoedema as a mental disease, and so it is in part of its symptomatology, in its torpor, dullness and dementia, but there are changes in hair, skin and other tissue as well as in the general bodily activity fully as important, while its cause is definitely a lowered activity of the thyroid gland.[5]

THE EVOLUTION OF PSYCHIATRIC ATTENTION

Since the mental diseases have relationships to diseases of the brain and diseases of the bodily organs, as well as to generalized pathological conditions such as arterio-sclerosis and drug intoxication, it becomes evident that biologically[6] they belong, as has been stated before, in quite different groups, and that consequently they cannot be, and are not of unitary character. Psychiatry is therefore a branch of medicine dealing in the main with disease phenomena, but *in its early days* it was interested mainly in mental phenomena, because these were the outstanding striking phenomena of the cases.

The transfer of psychiatric attention from mental symptoms to clinical and laboratory signs can be illustrated by the history of general paresis. A man develops ideas of grandeur, calls himself God, assumes lofty attitudes, attempts to kill those denying his divinity, becomes immoral and deteriorated—these are changes of such remarkable nature as to enlist Society, the Law and Medicine against him, and he is locked up as insane. Then in the course of generations an astute observer notices that the pupils are changed in such individuals, that they possess a peculiar immobility to light stimuli, and others observe that if one taps the patellar tendon in such cases that the knee jerks thus obtained are usually very lively, or in some cases absent. Thus there is added something of importance besides the mental phenomena which now interests the psychiatrist, viz., clinical neurological signs. Sometime later the development of post-mortem science discloses the fact that the brain of the patients of this kind shows decided changes, and also in patients showing quite

[5] Indeed it is well known that the lowered metabolism of myxoedema is the basic symptom and the mental symptoms are entirely secondary to this phenomenon.

[6] I am using the term biological in the wide sense that disease of various parts and from various biological sources (germs for example) is therefore a biological matter.

different symptoms, and there becomes built up a disease-entity called *general paresis*, and the psychiatrist is interested now, not as much in the mental pictures as in the brain changes that condition these pictures. Then there come rapid advances, because Medicine as a whole advances, and the psychiatrists' fighting line against this "mental disease" changes entirely in character since it is now learned that blood and spinal fluid show more definite evidence than the mental signs, are much more diagnostic and conclusive; that the cause of general paresis is syphilis, that its treatment is in the main the treatment of syphilis, and that this is independent of mental symptoms. Thus the mental symptoms of general paresis sink into relative unimportance, the status of general paresis as a mental disease medically becomes secondary to its relation to syphilis, a "venereal" disease, a disease involving arteries, liver, bones, joints, skin and mucous membranes. *In other words general paresis, a mental disease, becomes biologically nearer to a syphilitic infection of the liver than it does to another "mental disease," dementia praecox.*

I cite this evolution of our knowledge in the case of general paresis because it is the drift of things in psychiatry. True the psychiatrist now uses the work of the psychologist, but I venture to say he has given more to psychology than he has taken from it, even with the fine psychological contribution of the Binet Tests. But far more than he has need to be a psychologist he must be a pathologist, neurologist, and serologist—he must know about blood, urine, spinal fluid, endocrine glands, metabolism and the main data of medicine.

All this adds to the burden and folly of the term insanity[7] or any such unifying concept. Each disease must be established on biological grounds and studied separately. Cleared of the theological, mystical and legal alliances implied in the word insanity, psychiatry as a branch of biological medical science will make real and rapid headway.

[7] Nor does it do any good to substitute some other word to mean the same thing as insanity, such as psychosis which is now used. It is the implication of a unitary something, not the particular word, which is inimical to clear thinking.

CHAPTER II

A Few Words about Symptoms

Throughout this book, and especially in the case records cited, terms will occur which are in a sense the slang or argot of psychiatry. In the main these relate to mental symptoms, and in order to make clear the situation to the non-psychiatric reader it is necessary to add a short account of the meaning given these terms. This might be done in a glossary but with less satisfactory results.

DELUSIONS

The term delusion is used to indicate a false belief, one that is further removed from reality and truth than those beliefs which pass current as valid in the community. We cannot enter here into any discussion as to the nature of truth and though we may regard as false the beliefs of those differing from us in religion, politics, or morals, we do not regard them as insane if they correspond to ideas fairly prevalent in the community or having some historical or social basis. If a Southern Negro believes in Voodoo that is not necessarily a delusion; if a Harvard professor accepts such a belief he is "insane." One may have an enemy, and many of us do have enemies, but if an obscure citizen commences to believe that the Masons are organized against him and are seeking all manner of means to destroy or ruin him, that is a delusion. Delusions are divided into various groups, the most important being delusions of grandeur in which the patient imagines himself to be some high personage, even reaching the climax of absurdity in believing himself to be God; delusions of self-deprecation in which the reverse trend is noted, the patient believing himself to be the lowest of the low and accusing himself of all manner of crime; delusions of reference in which the patient believes that people talk about him in a way not at all likely to have occurred, that strangers notice him and point him out to one another wherever he goes; delusions of persecution which are exemplified in the idea that the Masons or the Catholics, or non-organized groups of persons are endeavoring to hurt or injure him in some extraordinary and

unlikely fashion; somatic delusions in which the patient may believe that he has no bowels or no heart, that the brains have been removed, that he has no genitals, that his lungs are decayed, etc.; delusions of jealousy, common in both male and female, in which unfounded and unreasonable accusations are made against the mate; nihilistic delusions—here the victim states that nothing exists, sun and moon are gone, he is dead or others are, etc.

HALLUCINATIONS

Hallucinations are those errors of the senses as a result of which something is perceived to be present which is not present. The most common are visual hallucinations (of animals, of supernatural creatures, of God, of enemies, etc.); auditory, in which voices of all kinds are heard; of smell, vile odors, gas, chloroform, etc.

ILLUSIONS

An illusion is the term given to a deception of the senses based upon a reality. That is, a patient may declare a chair to be an animal or mistake the appearance of those around him declaring them to be devils or ghosts, enemies in disguise, etc.

DEMENTIA

Dementia is a term used to imply that there is a failure in the intellectual processes, this failure usually taking the form of impaired judgment, loss of memory, failure of school knowledge, and sometimes used to include the loss of the esthetic and moral sense as well. The term dementia, of course, implies a *loss* and is in this respect different from the term feeblemindedness which implies that there has been no loss but that the individual has been deficient from the start. Some authors believe that the type of dementia differs in the various kinds of mental disease. This cannot be entered into here but I may say that I have never seen any real convincing demonstration that dementia, in itself, is distinctly different in various diseases.

THOUGHT DIFFICULTIES

Retardation means delay in the thought processes and is seen at its best in the depressed phases of the disease known as manic-depressive insanity. The term *blocking* is similar in its outer mani-

festations to retardation but has not the same mechanism at bottom, is believed to be due to splitting and inhibition of the thought processes, and is more commonly seen in dementia praecox. *Flight of ideas* is the type of association of ideas seen in the excited phases of manic depressive insanity and also in other diseases. The patient associates in a shallow manner, his mind being governed largely by sights and sounds of the outside world and it is indirect and rapid in its workings. The goal of the thought processes becomes lost and the patient's mind works in an irrelevant manner.

MOOD CHANGES

Mood or emotional changes are amongst the most important of the mental changes seen. These take three main forms. Euphoria means, in psychiatry, an abnormal feeling of well-being, an exaggerated happiness. *Exhilaration* carries the same idea plus excitement, talkativeness, etc. *Depression* means somewhat more than the same term in ordinary speech, it is hopelessness exaggerated often beyond belief. *Apathy* is perhaps not a mood change and implies a lack of emotion or at least a lack of emotional manifestation. This is common in dementia praecox and shows itself in the so-called "bench-type"; where the patient curled up on or under a bench apparently sees and hears nothing, and drools his days away.

CONDUCT CHANGE

Conduct change will of course be prominent in the mental diseases. Violence, homicide, suicide, immobility, need no explanation. "Psychomotor activity" means excitement, feverish actions, tireless running to and fro often for days and days as if fatigue were never felt. "Catatonia" is a term meaning many things, and at present a controversial term. Originally it meant a disease, then it became a part of the Kraepelinian triumph, dementia praecox, and now it has the status of a group of symptoms found in several diseases.[1] The principal symptoms of catatonia are first immobility with fixed attitudes, like a sort of "living picture" without grace, and in which the patient will maintain any attitude in which he may be placed by

[1] There is now a catatonic type of epidemic encephalitis. Urstein (204), Kirby (104), and others, have brought catatonia into the syndrome of other diseases, such as manic-depressive insanity, involution psychoses, etc.

the examiner. "Cerea flexibilita" is used to describe this type of motor plasticity. Such patients tend to repeat words, phrases, and movements in a curiously mechanical manner. Opposed to this passivity is, second, negativism, in which the catatonic shows a tendency to resist in a contrary manner whatever those in his environment try to get him to do—to stop still if they wish him to walk, to run if they wish him to stop, to close his mouth if they ask him to show his tongue, to refuse to eat or talk, etc.

The above are a few of the symptoms noted in mental diseases. For a fuller consideration I refer the reader to the text books on psychiatry. In the text books the meaning and significance of each symptom may be analyzed and traced to its psychological or physiological roots. Much of this is controversial, and this is not the place to enter into these disputes. I have aimed merely to make intelligible my use of the terms that will occur throughout this book.

CHAPTER III

Groups of the Mental Diseases[1]

We often hear of hereditary talents, hereditary vices, and hereditary virtues, but whoever will critically examine the evidence will find that we have no proof of their existence. The way in which they are commonly proved is in the highest degree illogical, the usual course being for writers to collect instances of some mental peculiarity found in a parent and in his child, and then to infer that the peculiarity was bequeathed. By this mode of reasoning we might demonstrate any proposition; since in all large fields of inquiry there are a sufficient number of empirical coincidences to make a plausible case in favor of whatever view a man chooses to advocate. But this is not the way in which truth is discovered; and we ought to inquire not only how many

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[1] It is not intended in this, and the following chapters, to discuss the field of psychiatry in its clinical aspects. Nor shall I take up all the mental diseases. In the first place some of the mental diseases, as for example, the traumatic psychoses are but few in number and naturally play no part in the discussion on heredity or inheritance. Further some of the subdivisions of various groups, such as Alzheimer's disease, seldom or never are diagnosed in life, and are only discovered on post-mortem examination. Dealing in large part with records often meagre, a fine (in the sense of a minute) division of the mental diseases, such as that of the American Psychiatric Association, would be of little value to me or the reader.

Those who wish to study the fundamental conceptions and the clinical details of psychiatry are recommended to the following works:

Kraepelin, E.: Psychiatrie. Eighth edition.
Bleuler: Psychiatria.
White: Outlines of Psychiatry.
Foundations of Psychiatry.
Buckley, Alfred C.: The Basis of Psychiatry, 1922 (an excellent, little known book).
Diefendorf, A. R.: Clinical Psychiatry, 1918.
Kraepelin: Dementia Praecox.
Bleuler: Schizophrenia.
Myer, Jelliffe and Hoch, Mendel, Krafft, Ebbing, Griesinger: Dementia Praecox.
Mandsley: (The Pathology of Mind) is valuable for the older conceptions of mental disease.
May, James V.: Mental Diseases, 1922. This last is an extremely valuable book because of the clear statement of the status of mental disease.

instances there are of hereditary talents, etc., but how many instances there are of such qualities not being hereditary. Until something of this sort is attempted we can know nothing about the matter inductively; while until physiology and chemistry are much more advanced we can know nothing about it deductively.

These considerations ought to prevent us from receiving statements (Taylor's Medical Jurisprudence pp. 664, 678) and many other books which positively affirm the existence of hereditary madness and hereditary suicide; and the same remark applies to hereditary disease.—*Buckle's History of Civilization in England.*

Psychiatry originally confined itself to those diseases in which "insanity" was a common ending. Originating as it did in the asylum, the asylum cases formed its subject matter and offered it its standards of thought. It is something as if one judged all mammals by elephants, and of these refused to study anything but the adults.

Today we know that there are far more non-asylum cases of mental disease than there are of those needing asylum care. Not only is this true when we consider only the diseases which are often committed to state hospitals but it becomes completely uncontrovertible when we include in the mental diseases the innumerable cases of character defect, neurasthenia, hysteria, etc. In other words the problems of mental disease reach almost into every home, and very certainly into every workshop, school, church and jail, and are part of the problem underlying such great social phenomena as poverty, crime, social conflict, religion and prostitution.[2]

What I wish to do, however imperfectly, is to discuss the biological problems underlying the various groups of the mental diseases. In order to do this I shall deal more with causes than with symptoms, since it is in their causes that we link together phenomena which are on their face different. And before going on with this analysis I wish to use as a paradigm which I think is in many respects relevant to the subject, the disease tuberculosis.

[2] Here I must protest against any assumption by psychiatrists that their special knowledge or science will enable them to solve the problems of poverty, prostitution, crime, economic conflict, etc. They who promise too much awake either suspicion or false expectations, both of which hurt their cause. Psychiatrists can help in the solution of these problems, or in their understanding, but their contribution touches only one face of the problems and not necessarily the important face. For example, in economic conflict they may be of help in an efficiency program, but they do not and cannot touch the vital matters of the relations between capital and labor.

PARADIGM OF TUBERCULOSIS

No one doubts that tuberculosis of the gastro-intestinal tract is essentially the same disease as tuberculosis of the lung. All the varying manifestations of tuberculosis are disregarded in the unifying concept of their common cause. The main symptom of tuberculosis of the larynx is loss of voice, "aphonia," and the main symptoms of tuberculosis of the knee-joint is loss of motion at the knee. Yet we do not class tuberculosis of the larynx with hysteria just because the latter sometimes has aphonia as a symptom, nor do we class tuberculosis of the knee joint with gonorrhoeal rheumatism because the latter also disturbs the motion of the knee joint. The diarrhoea and bloody flux of gastro-intestinal tuberculosis are not reasons for considering it a disease related to typhoid fever which also is manifest by diarrhoea and bloody flux. Biologically typhoid and tuberculosis are different diseases though they may coincide in some symptoms. The cough of pulmonary tuberculosis, the aphonia of laryngeal tuberculosis, the diarrhoea of gastro-intestinal tuberculosis, the scars of skin tuberculosis, or lupus, and the mental and neurological signs of meningeal tuberculosis do not prevent us from recognizing that we are dealing with one disease which has its foci in different locations and therefore disturbing different functions.

There is a point in the history of tuberculosis which is rather pertinent to the discussion of mental diseases. Up till Trudeau's time, in fact right up to the discovery of the tubercle bacillus by Koch, the main factor in the causation of the disease was held to be heredity. There were plenty of families found in which tuberculosis occurred "generation after generation," plenty of families found in which all or many of the brothers and sisters had the disease, the same kind of evidence we have today for the "inheritance of insanity." Just as we speak today of the psychopathic or neuropathic inheritance so our predecessors of past medical generations spoke of the "scrofulous type" or the lymphatic type, and made these types include everything from slenderness to fainting spells, including rickets (which we know now to be dietetic in origin), catarrhal affections of throat and nose (usually due to adenoid and tonsil hypertrophy), tetany (a defect in calcium metabolism), etc. With the discovery of the infecting organism as a cause of tuberculosis heredity received a knock-out punch; in fact every one now loudly proclaims that there is no heredity in tuberculosis but instead the

metaphysical "predisposition" is acclaimed, and still lingers to obfuscate intelligent thinking. What we really know is this, that the human being easily acquires tuberculosis under bad conditions of living, and that good conditions of living ward it off[3] that it is treatable by diet, rest, hygiene; that the environment causes it, in short, and that by control of the environment the disease can be eliminated in a remarkably successful way as is now being done.

Returning to the mental diseases, it is therefore proper to place them, biologically and logically, in groups according to known causes.

GENERAL PARESIS AND THE SYPHILITIC PSYCHOSES

For years general paresis (dementia paralytica) was not separated from the rest of the organic mental diseases. About the early and middle parts of the last century it began to occupy a separate and important part, but its cause was not at all suspected. It was due to the brilliant clinical insight of Fournier, and in lesser degree to Erb, that the cause of this disease, as well as of tabes dorsalis, was placed to the credit of syphilis. But even when it was in the main accepted that syphilis was the cause of the disease, its late development, the absence of the ordinary signs of syphilis, and its resistance to anti-syphilitic treatment led to the concept of parasyphilis or metasyphilis, conditions syphilitic in origin but not really syphilitic, a sort of modified result due to other factors. Then came the discovery of the Wassermann reaction, the study of the spinal fluid, and finally the discovery by Moore and Noguchi that the organism of syphilis, the spirocheta pallida, is present in the brains of paretics, and the status of general paresis as brain syphilis was fully established. Therefore general paresis is biologically a syphilitic disease, its relationship to syphilitic aortic aneurism, gumma of the liver, bone and skin syphilis is definite, whereas its biological relationship to dementia praecox for example or manic depressive insanity is only incidental.[4]

[3] The decline in tuberculosis as a major disease is well under way, and it is now freely predicted that in twenty-five years it will no longer rank as "one of the prime ministers of death." Some statisticians claim that the death rate from tuberculosis will again increase, but there is no evidence that this is coming to pass.

[4] Pilez (155) is the only psychiatrist of any standing who relates general paresis in an ancestor to dementia praecox in a descendant. He claims that

It is true that general paresis has often been declared to follow mild cases of syphilis, and the statements are made that it is not seen in oriental races though syphilis is prevalent in such races[5] and that there is in general a neuropathic background to general paresis. In my opinion these statements are mainly a lingering of the theories of general paresis that prevailed before the knowledge of syphilis as its cause came on the scene. It may be true that "mild" cases of syphilis end in general paresis but there may be a strain of the organism having a predilection for the brain[6] and it may also be true, as is often asserted, that in races in which syphilis has long been prevalent the skin, etc., manifestations are mild while the nervous manifestations are severe. As to the "Neuropathic" heredity of the general paretics this is obtained by discovering that they have "nervous" brothers, sisters, parents, uncles or aunts, or that these folks have headaches or are short tempered, that some are insane or feeble-minded, etc. Hardly any one could escape neuropathic heredity by the same criteria. "Without syphilis, no paresis" ends the discussion.[7]

Here enters an important fact. (*a*) The physical signs of general paresis range from very few departures from the normal to marked changes in the pupils (Arygll Robertson type), marked speech defect,

dementia praecox patients are in general the descendants of syphilitics. No one else has made this statement, and the physical examination, the history and the serology of dementia praecox are all against this astounding statement.

[5] Recent clinical studies of oriental peoples disprove this. (Lennox (120), William G.). It appears from this article that wherever careful studies amongst oriental peoples are done, the number of neuro-syphilitics rises immediately to occidental proportions.

[6] A. Marie and Levaditte make the claim that there is a neurotropic type of Spirocheta pallida.

"Psychotic or psychopathic taint does to some extent make a person more liable to general paralysis but only by inciting him to reckless indulgence of sexual appetite and so increasing his risks of syphilitic infection; the taint has no greater importance for the production of general paralysis than for the acquisition of syphilis. There are various indications that the exceptional proneness to general paralysis in certain families is due to some familial peculiarity of the physical defences against the spirochete, there is no indication that this peculiarity is in any way related to hereditary mental taint." Sidney J. Cole (29).

[7] "It appears, however, that the influence of individual peculiarities does not very materially modify the leading characteristics in mental disorders produced by noxious agents from without." Kreapelin (111).

tremor, clumsiness, increased or absent tendon reflexes. (*b*) The mental signs of the disease vary from the early character changes, the early "neurasthenia" to the typical grandiose dementia, the grinning, stuttering, trembling demented patient who proclaims himself as "f-f-feeling f-f-fine" and the greatest personage in every way from sex power to mathematical knowledge, *but there occur in addition mental disease sypmtoms not to be distinguished from dementia praecox or manic-depressive insanity* except by the so-called biological tests. In other words the same physical cause is capable of producing a wide variety of mental signs and symptoms. (*c*) But the biological signs are remarkably constant.

1. The Wassermann reaction in the blood is rarely absent.
2. The Wassermann reaction in the spinal fluid is rarely absent.
3. Almost invariably the protein content of the spinal fluid is increased.
4. This is also true of the cells that are found in the spinal fluid—they are increased and moreover tend to be of the same type in every case (lymphocytes, plasma cells).
5. The gold sol changes (Langes' test) is more nearly uniform in general paresis than in any other disease where such changes occur. And all this is true almost regardless of the mental or physical signs, in the early stages or the late stages. I repeat what I have said before, the attention of the psychiatrist has shifted from the mental signs of general paresis to the biological (or serological) signs because the former are incidental as compared with the latter. When it is remembered that of the admissions to the State Hospitals of the United States over 11 per cent[8] are general paresis, will be realized that here is a great field where heredity plays no important rôle as a causative agent, and where the environment as an infective agent is *the* factor.[9]

How does the general paresis of an ancestor affect descendants? The answer may be tersely put, *as syphilis in general affects the descendants.* The work of the Solomons may be cited as typical of the results found by most of the investigators.

[8] May, p. 307.

[9] Those interested in the symptoms, pathology, and laboratory signs of general paralysis and the syphilitic psychoses will find a discussion in Kraepelin (108), Nonne (144), Southard and Solomon (180), May (127), Alzheimer (5) and others (see bibliography).

In their excellent book "Syphilis of the Innocent" H. C. and M. H. Solomon (178) have summarized the familial effects of late syphilis as follows:

A summary of our findings in this study of a consecutive series of the families of late syphilitics shows:

1. *The family of the late syphilitic abounds with evidence of syphilitic damage.*

2. At least one-fifth of the families of syphilitics have one or more syphilitic members in addition to the original patient.

3. Between one-third and one-fourth of the families of syphilitics have never given birth to a living child. This is much larger than the percentage obtained from the study of a large group of New England families taken at random. Here it is shown that only one-tenth were childless.

4. More than one-third of the families of syphilitics have accidents to pregnancies, namely, abortions, miscarriages or stillbirths.

5. The birthrate in syphilitic families is 2.05 per family whereas the birth rate in the New England families mentioned above is 3.8 per family or almost twice as high.

6. *Over one-half of the families show defects as to children (sterility, accidents to pregnancies, and syphilitic children).*

7. Only one-third of the families show no defect as to children or Wassermann reaction in spouse.

8. About one-fifth of the individuals examined show a positive Wassermann reaction; more of these are spouses than children.

9. Between one-fourth and one-third of the spouses examined show syphilitic involvement.

10. *Between 1 in 12 and 1 in 6 of the children examined show syphilitic involvement.*

11. One-fifth of all children born alive in syphilitic families were dead at the time the families were examined. This does not differ materially from the general average in the community.

12. One-fifth of the pregnancies are abortions, miscarriages, or stillbirths, as compared with less than one-tenth of the pregnancies in non-syphilitic families.

13. The average number of pregnancies per family is 2.58 compared with 3.88, 4.43, and 5.51 in non-syphilitic families.

14. There are 3.52 still births per 100 live births in the syphilitic families, as compared with 3.79 reported by the Massachusetts Census study of non-syphilitic families. This shows no very marked difference.

15. *A syphilitic is a syphilitic, whether his disease is general paresis, cerebrospinal syphilis, or visceral syphilis without involvement of the central nervous system, and the problems affecting his family are the same in any case.*

These conclusions of the Solomons do not fundamentally differ from those found in the literature wherever the problem has been closely and clinically studied. The reader is referred to the work of F. v. Rohden, who has summarized the German work done on this

subject and also made an independent study of excellent type. The conclusions of von Rohden are that the descendents of patients suffering from tabes or general paresis are syphilitic to a very large degree, and that this syphilitic involvement is a matter of syphilis of the mucous membranes, arteries and nervous system, just as it is in the acquired form. von Rohden (159) points out that the children born in the early stages of a syphilitic infection of the parent are very much more likely to be diseased than those born in the later period of the infection. It would be, therefore, of great importance to study the children of non-neurologically affected syphilitics, and von Rohden believes that these would show even a higher degree of injury than the descendants of paretic or tabetic. He cites in detail the types of injuries found, and emphasizes injury found in the descendants. *He emphasizes that there is no original or acquired predisposition to general paresis, that syphilis and syphilis only is the cause of general paresis, and that hereditary factors play no important part in the acquirement of congential syphilis, the only factor of importance being the syphilis of the ancestor.* von Rohden's work emphasizes the point taken throughout this book that where a definite and direct etiological cause is found for a condition hereditary factors are of no essential importance, and it is clinical wisdom not to be oversubtle in dealing with the more or less hypothetical predisposition.[10]

THE BIOLOGICAL RELATIONSHIPS OF OTHER ORGANIC DISEASES

General paresis is only one of a number of "organic" diseases of the brain that cause mental disease. Though it is the most spectacu-

[10] Those interested in the matter of the descendants of syphilitics are referred to the following writers:

Fournier: Syphilis secondaire tardive. Paris, 1906, Berlin, 1909.
Haskell: Familial syphilitic infection in general paresis. Jour. Amer. Med. Assoc., 64, 1914.
Hauptmann.: Serol. Intersuchungen von Familien syphilogener Nervenkranker. Zeitschr. f. d. gest. Neur. u. Psych., 8, 1911.
Hochsinger: Die gesundheitlichen Lebensschicksale erbsyphilitischer Kinder. Wiener klin. Wochenschr., 1911, Nr. 24/25.
Hübner: Über kongenitale Lues. Archiv f. Psych. 57, 1917.
Nonne: Über Syphilis congenita in 3. Generation. Neurol. Centralbl., 1916, Nr. 4, Ref. 22.
Raven: Klin. und serol. Untersuchengen an den Familien von 117 syphilogenen Nerven krankheit. Deutsche Zeitschr. f. Nervenheilk., 51, 1914.
Scharcherl: Über Luetikerfamilien. Jahrb. f. Psych., 36, 1914.

lar and strikes down its victim at the most fruitful period of his life, yet in actual numbers it ranks as less important than other diseases of the brain. Thus in 1920, the first admissions to the Massachussetts State Hospitals for the Insane totaled 2819, and of that number 264 or about one-ninth, were due to cerebral arterio-sclerosis. This is exclusive of the so-called senile dementia cases (to be discussed later) which in at least a large proportion are really organic in the sense that they are due to changes in the brain substance.

The problem of cerebral arterio-sclerosis, which is thus so important in the rôle it plays in "insanity," is distinctly not mental.[11] It is usually a problem of infection, kidney disease, heart disease and general arterio-sclerosis. It is found *more commonly*, if anything, in the ancestors of the normal than the ancestors of the "insane."[12] It is exceedingly common, and sometimes takes the form of hemiplegia, a paralysis of half of the body, due either to the rupture of a cerebral blood vessel, or to its closure by embolus or thrombosis. Commitment to an insane hospital takes place only when the lesions are more extensive than usual or where the social situation makes it difficult, impossible, or distasteful for the patient to be cared for at home. The mental symptoms are usually those of dementia and dullness, but there may be excitement and even hallucinations and delusions of all kinds. *These mental symptoms are but a small part of the clinical picture, which is dominated by the condition of the heart, kidneys and arteries.* This form of mental disease is thus, biologically and medically, a part of the problem of arterio-sclerosis, and has its roots, not in heredity, but in infection, wrong diet, stress and strain of life, lack of exercise and whatever it is that is back of arterio-sclerosis. True one sees many cases in families but then, the social hygienic and environmental conditions of the various members of families are apt to be the same, and this is a far more rational explanation than to fall back on "heredity." *In fact I suspect an inferiority complex in the ready use of heredity as explanatory of many conditions—because they do not respond to our present medical treatment we throw off the feeling of inferiority that their existence forces*

[11] It is true that the liability of the arteries to wear out early has been ascribed to heredity on a rather slim basis of actual cases. But this liability to arterio-sclerosis is a different liability than that to mental disease. See May for a clear discussion of the literature as a whole.

[12] See pages 105 ff; Diem (46), Koller (106), Jolly (99).

on us, and take refuge in heredity as a cause, since no one can rationally expect us to eliminate "heredity."

In the chapter on family studies I shall present some cases of mental disease that have a later generation succeeded cases of organic brain disease in an ancestor. At the present let me flatly state that there is no good evidence that cerebral arterial disease, as a cause of mental disease, has any relation to heredity in either the ancestors or descendants of the patient. Very few indeed of the published cases of family mental disease concern arteriosclerotic mental disease.

MISCELLANEOUS ORGANIC DISEASE OF THE BRAIN

Under this heading is included various diseases, brain tumor, paralysis agitans, tubercular or other forms of meningitis, multiple sclerosis, and other conditions which under certain circumstances bring about enough mental disturbance as to bring the patient to the Insane Hospital. In a general way such patients do not "belong" in this type of hospital, and the majority of such sufferers are in the chronic non-insane hospitals, the alms-houses, etc. Only when they become too disturbed to be cared for elsewhere, or where there is a mistake in diagnosis, do they reach the Insane Hospital.

Obviously heredity is of small importance in such diseases if it plays any rôle at all. Tumor of the brain is part of the problem of neoplasm, a problem we are far from thoroughly understanding, and in which the theories of causation vary from the idea of infection to the more generally accepted belief that something sets agoing "cell anarchy" so that certain cells start to grow and multiply without regard to the welfare of the organism. The problem in multiple sclerosis now shows signs of being solved on the basis of chronic infection and the mysterious "idiopathic degeneration" hitherto invoked as the cause, will undoubtedly give way to clearer concepts. Multiple sclerosis does not run in families, and rarely gives rise to mental disease. The problem in paralysis agitans[13] is now well defined—degenerations take place in the lenticular nuclei at the base of the brain, and we now know that a peculiar "lenticular syndrome" follows epidemic encephalitis. Everything points toward infection as a basis for this disease

[13] A series of workers carrying on brilliant researches have brought us to our present understanding of this and related diseases. I have in mind especially Wilson (217) and Ramsay Hunt (88).

type. As the figures of Koller, Diem, and Jolly show, organic disease of the brain is probably more common in the ancestors of the normal than in the ancestors of the abnormal, and all the logic of the situation as well as the drift of knowledge is toward the conception that each of the great organic diseases is a problem by itself, and without definite relation to heredity or to other brain disease.

THE TOXIC PSYCHOSES—ALCOHOL, DRUGS, ETC.

From the very earliest days of which we have any record, and in every civilization however primitive, men have sought pleasure or relief through some intoxicant or other.[14] In our civilization alcohol in its varied forms has been the chief medicine through which Euphoria or her less joyous but still welcome sister Anaesthesia were won. In the oriental civilizations, and in recent generations invading our occidental habits, opium and its derivatives, hashish or Cannabis indica have softened the rigors of life, given a pleasure or forgetfulness for which some have paid a penalty in mental disease.

ALCOHOL AND MENTAL DISEASES, THE RELATION TO HEREDITY

A few words are necessary in description of the common alcoholic mental diseases.

1. There is first of all drunkenness, which is a state reached by anybody who drinks to excess, and characterized by a wide variety of symptoms ranging from foolish and criminal conduct to unconsciousness, ranging, to speak physiologically, from a loss of inhibition to a complete loss of function. Though people differ widely in the amount necessary to get them drunk, there exists no doubt that any one may become drunk. There is therefore merely a difference of degree in the resistance to alcohol in this matter. These cases never or rarely reach the insane hospital.

2. There is the very rare "pathological intoxication," associated with periods of amnesia after drinking. The alcoholized person, in

[14] "In the tragic conflict between what he has been taught to desire, and what he is allowed to get, man has found in alcohol a sinister, but effective peace maker, a means of securing, for however short a time, some way out of the prison house of Reality back to the Golden Age. Some too ignorant, some too cowardly, or perhaps too brave to find a release there" (Trotter (200), The Herd).

these cases, may set fire or carry on complicated procedures, without any later remembrance. In this respect the condition notably resembles the fugues of hysteria. There are too few such cases recorded to make any conclusions.

3. There is the common condition of "delirium tremens." Necessary for its production is first prolonged drinking, and second there often occurs as an exciting cause, a sudden injury or an acute illness. Whether or not there needs be a "psychopathic" temperament is a question which I defer discussing now, but there surely needs to be chronic heavy drinking. Delirium tremens presents a clinical picture not unlike the delirious states found in the acute infections like typhoid or pneumonia. The patient is markedly hallucinated, mainly in the visual field, is confused to an extreme degree, is tremulous, often has an elevated temperature, and there is frequently a serious prognosis as to life. Delirium tremens is rarely cared for in the insane hospitals because the disease is sudden in onset and the recovery prompt in the majority of cases.

It is probable that not every chronic drinker is a candidate for delirium tremens. Does that mean that we are to consider that in every case of delirum tremens there is a neuropathic liability allied to a general liability to mental or nervous disease? It would, it seems to me, be as logical to assume that every patient with delirium, e.g., in penumonia, is therefore neuropathic. It is true that peculiar, odd, and eccentric people, as well as the feeble-minded and the insane, develop delirium tremens, but it is also true that strong, robust and apparently normal men develop this disease under the essential condition, of course, of heavy drinking and the common associated conditions of injury, or acute sickness.

4. Alcoholic hallucinosis: This is a fairly common mental disease due to alcohol, and marked by the appearance of delusions, usually of persecution and based apparently upon hallucinations of hearing. Usually there is little or no confusion, the patient is "clear" in so far as memory, orientation, and the ordinary criteria are concerned. In a greater or lesser period of time the hallucinations and delusions fade and disappear and never reappear *unless* the patient begins to drink heavily again.

In many cases it is difficult to differentiate this disease from certain forms of dementia praecox, but time and abstinence from alcohol nearly always furnish the proof.

We may say that relatively few drinkers develop alcoholic hallucinosis, and there seems to be a special predisposition to this mental disease. Is it a hereditary predisposition? The family studies I will later cite show no cases of alcoholic hallucinosis occurring with paranoid disease of other origin, and very few cases where alcohol hallucinosis occurs in brothers or sisters, or where a father or mother with alcoholic hallucinosis has children with like or dissimilar mental diseases. But my cases are far too few from which to draw any conclusion, and undoubtedly in all hallucinoses there are psychopathic factors at work in addition to the alcohol.[15] These factors may make up a constitutional peculiarity which need have no "origin" in germ-plasm. That is, there seems to be an *individual* peculiarity, but this does not mean a peculiarity which is *hereditary* or transmitted from generation to generation. We must sharply differentiate individual peculiarity from hereditary peculiarity.

5. Allied to alcoholic hallucinosis is alcoholic paranoia, a mental state mainly characterized by delusions of jealousy, directed against the mate, usually the wife. This disease lasts during the heavy drinking, and disappears with abstinence. Relatively rare, it is sometimes explained on the psychological basis but the drinker, partly impotent but with inflamed desire, is repugnant to his wife, is repulsed, and builds up as a consequence an explanatory delusion that she is unfaithful. What I have said in regard to alcoholic hallucinosis applies to alcoholic paranoia, viz., that some personal peculiarity *seems* necessary in addition to the alcoholism, second, that it does not occur in family groups, though alcoholism often does, third, that it does not seem to "breed" other mental diseases.

Here it is pertinent to make a few remarks about logic and medicine. If a large number of people are exposed to a contagion and only a few develop a disease as a result it is often assumed on the ground of logic, that there existed a special predisposition, an innate weakness, or susceptibility in the case of those who become sick. But this does not take into account the *fluctuations* in all organic functions—resistance to disease, for example—it does not take into account that if there is fluctuation in resistance, *coincidence of infection with the moment of lowered resistance, coincidence of infection with the moment of heightened resistance may mean disease or non-*

[15] See Stocker (190).

disease in the case of two individuals *not* essentially different. Since the complex of circumstances is so intricate as to defy disentanglement, it is safer to consider only the factors that are inevitable as causative. The only inevitable factor in general paresis is syphilis, even though only 5 per cent of all infected with syphilis acquire the disease and the only inevitable factor in the alcoholic psychoses we have thus far considered is alcohol. In certain people strawberries may cause "hives," diarrhoea or asthma, but we do not believe that any profound hereditary mechanism is behind the peculiar reactions. Amongst the immense number of heavy users of alcoholics in our population there is a universal liability to get drunk, a lesser liability to delirium tremens, and a still lesser liability to hallucinosis and paranoia; there is also a liability to heart disease, fat deposition, possible cirrhosis of the liver, and most certainly gastritis.

It is a cardinal observation in pharmacology that idiosyncrasy of a non-hereditary type plays an important rôle in dosage. That is to say, while the average dose of morphine is one-quarter of a grain there are people to whom such a dose acts as a tremendous poison and others on whom it has no effect whatsoever. So with bromides, some individuals are made drowsy by 10 grains of sodium bromide, others get a severe rash, and still others find it has a salty taste but has no constitutional effects whatever. Now we do not find a neuropathic basis for the resistance to bromides or the resistance to morphine, and I see no special reason why the resistance to alcohol should be made so significant. *The reason the resistance to alcohol has been made so important is that the use of alcohol has become a great social controversy and feeling has been awakened which is inimical to clear thinking.*[16]

At this point there are two interesting questions that come up which have received completely divergent answers from the investigations. Who drink excessively, is heavy drinking a sign of abnormal mental character, and related to mental diseases as a whole? Koller (106) and Diem (46), investigating the heredity of the "sane and insane" (a bad division of mankind but having some practical value) found that alcoholism occurred twice as frequently among the *parents* of the insane as among the parents of the sane (Koller), one

[16] May's (127) pertinent remark "With the advent of prohibition the alcoholic psychoses have become a matter of little more than historical interest" (p. 362) shows what really causes the alcoholic psychoses.

and a half times as frequently according to Diem. They report the curious result that in the history of *other relatives* (grandparents, brothers, sisters, uncles, etc.) alcoholism appears more frequently amongst these relatives of the sane than in those of the insane.

Most of medical opinion is back of the belief that there is a factor of psychiatric or psychopathic nature when an individual drinks heavily. For example, Lambert (118) starts his article on alcoholism "An unstable nervous system is the fundamental basis on which habitual alcoholic excesses develop. There is the weakness of will, the tendency to over-indulge, the lack of self control. . . . ." Just what an unstable nervous system is Lambert does not tell us and he neglects the fact that men with very firm will in regard to other matters have been heavy drinkers and that women who drink much less than men are not thereby of firmer will. Nor are we to assume that the Jews who drink little (or not often to excess) are of firm will or possess a stable nervous system. In fact will is no unit, and a man who can resist the lure of alcohol may be extremely vulnerable to sex, or may not resist the temptations of the table, or may succumb to Lady Nicotine. Why it is neuropathic to desire alcohol to excess and not neuropathic to smoke or eat incessantly is somewhat of a puzzle to me. Then Lambert goes on practically to invalidate his above quoted statement for he declares that the poor seek relief in alcohol from overwork and insufficient food, and that professions in which irregular hours are the rule furnish more drinkers than do those of regular hours, but also men who work hard and out-of-doors drink heavily—teamster, longshoremen, stokers, masons, iron-workers—surely not a list of neuropathic occupations!

In fact there are "neuropathic" people who drink to excess especially the chronically or episodically gloomy, and the depressed mental states are often ushered in by a bout which may mask the essential melancholia of the patient. But also as many, or more, of the "neuropathic" shun alcohol, hate it, cannot drink it. It is probable that social pressure, habit formation, ill health, type of occupation, tradition, and idiosyncrasy of the same kind that we find in relation to all drugs are back of much alcoholism. In times past a very large proportion of the men of Western Europe were alcoholics by our present standard but there is no one who seriously believes there was more neuropathy then. The American Irish drink ten times (or more) as heavily as the American Jews but where is the

psychiatrist of any experience who believes that the former are proportionately more "neuropathic" than the latter? Though the Jewish alcoholic is rare, and the Jewish alcoholic psychosis still rarer, the race furnishes its full proportion of dementia praecox, neurasthenia, epilepsy, and "feeble-mindedness" to name but a few conditions. In fact we may conclude that like the poor whose main trouble is their poverty, the alcoholic's main defect is his alcoholism.

This brings me to the much more important question. "Does the alcoholism of the parent adversely affect his offspring? Is it responsible for mental disease in the case of the descendant?" To this question the traditional answer of most of medicine has been an unqualified "Yes! Alcoholism in the parent is in part at least responsible for feeble-mindedness, epilepsy, criminality and insanity." This is the statement that in one form or another forms part of the stock solutions of psychiatry. Since the situation in regard to alcohol is that of a bitter moral and legal controversy, in which many are entered whose zeal and earnestness outruns by far their knowledge and candor, there arose the deep conviction that alcohol *ought* to cause "degeneracy" and that therefore it did.[17]

No one can deny that alcoholism brings social situations that are bad for the offspring, that it emphasizes and enhances the evils of poverty, ignorance, and crime. This is a far different matter than to state that it injures the unborn, and that in the alcoholic excess of the parent is the cause of the mental defect or disease of the child. An important group of investigators, the associates of Karl Pearson, have made a statistical study of the children of alcoholics and have come to the surprising and revolutionary conclusions that

1. The mean weight and height of children of sober parents is very slightly greater (when mathematically corrected) than those of the alcoholic.

2. The general health of the children of alcoholic parents appears on the whole slightly better than the health of the children of sober parents. There are fewer delicate children and in a most marked way fewer cases of tuberculosis and epilepsy!

3. Parental alcoholism is not the source of mental defect in offspring.

4. "To sum up then, no marked relationship exists between the intelligence, physique or disease of offspring and parental alcoholism

[17] Elderton and Pearson (47).

in any of the categories investigated. It is needless to say that we do not attribute this to the alcohol but to certain physical and possible mental characters which appear to be associated with the tendency to alcohol." (What is here implied and in fact the authors state, is that the alcoholic parent is better physically and mentally than the non-alcoholic!)

This astonishing conclusion is in part contradicted by two later studies emanating from the same group. In Eugenics Laboratory Memoirs XIV the results of a study of female alcoholics in reformatories is given in which the conclusion is reached that mental defects and alcoholism are correlated, and in a still later study by David Heron he shows a very high proportion of inebriates with mental defects.[18]

All of which shows that expert statisticians can prove contraries and that statistics can prove nothing, if the data are inaccurately gathered. For example, in the Eugenics Laboratory Memoirs XIV, dealing with women in the reformatories, the criteria for judg-

[18] In another memoir the likeness of husband and wife in biological qualities is studied through a statistical analysis of their age at death as recorded on tombstones! This does not take into account that husband and wife usually eat the same food, sleep in the same bed, are housed in the same building, and pass infection and worry to each other. I do not understand statistics, and this is probably why I regard the work of the English biometric school as a fine exercise in ingenuity, but with no real utility to psychiatry. Intensive individual study must be one arm of clinical science, and the other is experiment. When these arms gather data then statistical science may step in to interpret them. Until that time the statistician is like a color-blind man matching colors. Only by luck will he reach a correct conclusion.

For a full account of this controversy read the works of Ch. Fere (53), Horsley (86), Forel (57), the Eugenics Memoirs dealing with alcohol, nos. 10, 13, 14, 17, and especially Holmes (85) on the "Trend of the Race." Holmes gives a very impartial review of the evidence and literature. He cites Stocker whose work on the alcoholic psychoses is a classic to the effect that the inebriate is such because he is abnormal by birth. On the other hand the statistical work of Lewis and Sullivan shows that in England the rural communities have more pauperism and insanity, and less alcoholism than the cities and industrial centers, which would tend to show that industrial and social conditions play more of a part in alcoholism than any innate trend.

If one is interested in the psychology of controversy let him read J. M. Keyne, Journal of the Royal Statistical Society, July, 1910, volume 73, page 769, in answer to the work of Karl Pearson, and then read the answer made by Pearson to these gentlemen and to Sir Victor Horsely and Dr. Mary Sturge. There is rancor in every line.

ing mental defect are fit to make a psychiatrist weep. The mentally defective are those women whom a reformatory physician sets down as defective, not on any system of tests or study, but if they are (1) eccentric, (2) silly, (3) dull, (4) (ye gods) senile, (5) or subject to periodical paroxysms of ungovernable temper! The very defective are the congenitally imbecile (we are not told how that conclusion is reached), degenerate (sexually? if so, Socrates was mentally defective) and the epileptic. Then they are judged sick or well according to whether they are fit for hard work or not!

This does not alter the fact that medical men and prohibitionists have been naïve in their linking of alcoholism in the parent with the mental disease of the child. Until we know the cause of dementia praecox in a definite manner we cannot say that the fact that a parent was alcoholic had anything to do with it. In fact prohibitionists also have dementia praecox descendants. If there is more epilepsy in the descendants of the alcoholic (which I doubt) it is perhaps because the wives of alcoholics have less skilful obstetricians in attendance and their children are more liable to injury at birth, or the children receive poor food and have less attention paid to the bowels, or fall more frequently downstairs, or live where infection is more rife, all of which are causes of epilepsy, but to which the alcohol stands only as a *social*, non-biological, predisposing cause.

This may be said of alcoholism aside from the mental diseases due to alcohol.[19] A certain number of alcoholics are such because their mental troubles work themselves out in alcoholism. Such are certain of the periodic drinkers and the kind of drinking done by the abnormally depressed and seclusive. Others are psychiatric and incidentally alcoholic. One can understand that a feeble-minded man, especially of the hobo type might more easily become alcoholic than the non-feebleminded owing to the nature of the social station occupied, and the friendliness of the saloon to the derelict; and the same is true of the prostitute whose mentality may be of low type but whose environment is absolutely inimical to sobriety. Against these two

[19] At this point I must again refer the reader to the brilliant discussion of Holmes (85) on the relation of alcoholism to heredity and to mental disorders of all kinds. As one reads the articles he cites, and his own discussion, the difficulty of drawing any conclusions becomes evident. Culture and constitution, environment and heredity, tradition and germplasm mingle their effect especially in relation to alcohol, its use and abuse, in a Gordian knot no Alexander can cut.

groups of the abnormal alcoholic is in my opinion to be weighed a very much larger group (or groups) of alcoholics, whose alcoholism is *mainly environmental* or due to personal idiosyncrasy not at all psychiatric. That is, an abnormal fondness for alcohol may and does represent an isolated peculiarity not standing in any relation to mental disease, just as a partiality for sweets or a love of tobacco is not necessarily psycho-pathological. When we unify a trait into which so many components, social, biological, and psychological enter, we are guilty of an over-simplification which finally brings confusion. Therefore it is quite likely that the alcoholism of a parent *may* be and often is of a type to be considered in the heredity of a patient with mental disease. In the majority of cases I venture to say it is not of such type.

Can the drinking act as a toxic influence, cause prenatal injury? I shall refer to the work done in this field in detail later on, but at present the following will suffice:

Many medical writers have emphasized germplasm injury by toxic agents, and especially by alcohol. Note especially Forel (57), Hodge, Hoppe, Simmonds, Bertholet (11), Lenz (121), Horsly (86), Binzwanger (14), etc. See also Ch. Fére for a full account of the earliest writers.

As representative of this group of writers I cite August Forel (58). This distinguished physician lays down the law of blastophoria, which briefly is that the texture of the germinal cells may be injured and this injury is inheritable. Alcoholic blastophoria is a "Hauptquelle" (prime source) of degeneracy.

He says that in acute alcoholism the poison quickly reaches the seminal fluid cells and injures them in function just as it does the brain. He cites the changes in chronic drinking, the injury caused in testicle and ovary. He cites also experiments which anticipated those of Stockard, those of Combernale 1888, which show that not only do the immediate descendants suffer but that in the third generation the signs of degeneracy were greater than in the second generation. In other words a toxic influence sets agoing a progressive race degeneration.

He also cites Laitinen, 1909, whose alcoholic experiments indicate that (*a*) the drug in animals lowers resistance to toxins (this is well known in man in relation to pneumonia); (*b*) lessens haemolytic blood power; (*c*) *lowers vitality and growth of descendants.*

He answers Wilfred Paret whose dictum is "alcohol wipes out the canaille" by the counter-statement "it creates more canaille."

The brilliant experimental work that has been done need not be discussed at this point. What needs emphasis is this which so far as I know has not been hitherto considered: *that germplasms are not all alike in their resistance to toxins, any more than other tissues are.* In other words the germplasm of some persons may be injured by alcohol though the germplasm of the majority may easily pass through the ordeal without injury, as one man may get drunk on a glass of whiskey while another drinks a quart without definite or noticeable injury, just as some are poisoned by fish, or this or that protein, which to the majority are forms of nourishment. *The wide variability of the human being is an outstanding factor in all discussions on human problems, and it does not seem improbable that this extends to the resistance of the germplasm.*

OTHER TOXIC OR DRUG PSYCHOSES[20]

These include the mental diseases due to cocaine, morphine, heroin, and other narcotics. Only a small percentage of rather acute mental disease is caused by these agents (less than 0.25 per cent, May, p. 376) and what has been said of alcohol applies to these drugs, i.e., in some cases we deal with an individual of peculiar type, in many others we deal with those whom environment, social and economic conditions have forced into the drug habit. In some cases a chronic disease, such as locomotor ataxia or gastric ulcer is responsible for the formation of the habits, in others a painful social position (prostitution, criminality) brings about the use of drugs. It is of course to be emphasized that there are numberless cases of the drug habit to one of drug mental disease. As yet "prohibition" has not increased the drug psychoses though it may have increased the number of drug habituées.

TOXIC EXHAUSTIVE MENTAL DISEASE

A small group of mental diseases is the so-called toxic exhaustive psychoses, mental diseases following infective diseases, pregnancy, occurring in the course of tuberculosis, cancer, diabetes, etc. These

[20] Report on Morphinism to the Municipal Court of Boston. C. Edward Sandoz (169). I refer the reader to the above mentioned article for a fine account of the drug addict and a compendious literature.

conditions are marked by confusion, illusions and hallucinations, often by depressed delusions, and there is usually recovery from the mental state if the toxic condition in itself does not cause death. Though "heredity" is often stated to be a predisposing cause it is of the vague kind involved by a dozen different conditions such as alcoholism, crime, "insanity," (without definition of type), nervousness, cancer, tuberculosis, etc., and without regard to the nearness of the related person. I know of no definite studies carried on to establish the rôle of hereditary factors in the toxic exhaustive conditions, and my own opinion is that the *main* cause is the infection or toxic condition.

(It is of course true that mental diseases arise during pregnancy, cancer, diabetes, etc., especially the former. This is especially true of dementia praecox and manic depressive insanity, and in these diseases hereditary factors are of importance. These are not, however, included in the toxic exhaustive psychoses.)

PELLAGRA

Of great importance is the disease pellagra. Pellagra is endemic in Italy, Spain, Greece, Egypt, the southern United States, and southern France. The most of the opinion concerning pellagra states that it originates somehow in the dietic conditions of these parts of the world, and opinion has varied from the spoiled corn theory of Lombroso to the absence of milk, fresh meats and excessive carbohydrate diet of Goldenberger, with much stress laid upon the heat of the sun as a factor. The disease is characterized by (*a*) roughening and reddening of the skin in various parts of the body especially the back of hands and wrists, face and chest; (*b*) diarrhoea, and (*c*) a dementia. Death ensues in a fairly large percentage of cases, recovery often occurs, but the disease is apt to recur. There is a seasonal incidence and pathologically there is very marked acute change in the nerve cells of the brain and cord, as well as inflammation of the gastro-intestinal tract.

That the disease depends upon dietetic disorder is an old conception. "More than half a century ago Roussel on the basis of his own observations and the experience of others assigned to diet and especially to a milk diet the place of first importance."[21] As

[21] Goldberger's (67) articles give a very complete account of the literature. See also Sandy (170)

Goldberger points out this correct therapeutics was overthrown on the great authority of Lombroso whose distinguished career is mainly marked by great errors which his persuasive and powerful pen forced on the world. But even he believed that some dietetic intoxication caused the disease. The work of Goldenberger and his associates as well as those of Wilson, Enright, and others has been to establish a dietetic deficiency of some kind as causative, though other workers have found (Siler, Garrison, McNeal (67)) that bad sanitation in regard to the disposal of excreta was important. However, it is certain that this important disease, called a mental disease though its other symptoms are more important, is essentially environmental in its origin, and to be prevented by good hygiene and proper diet.

It is interesting to observe that Davenport and Muncey make a gallant effort to link up the disease with heredity in order to substantiate the Davenport idea that all mental diseases (and many other diseases) are a unit, due to a hereditary unit deficiency. Though Davenport cannot quite find a direct heredity he thinks there are constitutional factors, which determine the cause of the disease, because certain families have more of Pellagra than others, and pellagrins vary in the type of disease. All of which may be true and yet be of no hereditary importance since families usually have the same diet as well as the same heredity. His associate Muncey ferrets out a predisposition because some pellagrin families seem to have a good deal of gastro-intestinal disease of other kinds. Since pellagra is intimately associated with the causes of gastro-intestinal tract disease—very bad hygiene and poverty, there is no need to invoke other cause.[22]

[22] Statistics of pellagra in Spartanburg County. The Society for Experimental Biology and Medicine. Proceedings 12, 1914–15, page 42–43. (From the Robert M. Thompson Pellagra Commission of the New York Post-Graduate Medical School and Hospital.)

"Up to September 15, 1914, we have recorded about 1165 cases of pellagra, which have been recognized in Spartanburg County, S. C., the large bulk of them since 1910. The population of this county in 1910 was 84,000. The comparative study of the distribution of that portion of these cases recorded up to the end of 1913, in respect to geographical location, race, age, sex and occupation, has shown the disease to be most prevalent in the larger centers of population and especially in the cotton-mill villages. Pellagra has been about three times as prevalent among the white population as in the negroes. It was

OTHER TOXIC EXHAUSTIVE PSYCHOSES

Psychoses dependent upon bodily disease constitute about 4 per cent of the admissions to Massachusetts State Hospitals. They are, however, of so many origins that I content myself with merely enumerating a few causes of these admissions: infectious diseases, post-infectious states, exhaustion, cardio-renal disease, metabolic disorders (diabetes, cancer), endocrinal diseases. The mental states range from delirium to temporary dementia. Even the most fervent enthusiasts for heredity claim but little for this as cause in these diseases.

MENTAL DISEASES OF ENDOCRINAL ORIGIN

We are not yet at that place where we can safely discuss the rôle played by heredity in those mental diseases of endocrinal origin.[23] In the first place they play as yet a very small rôle in psychiatry, i.e., independent of speculation.

While some "endocrinologists" find an endocrinological origin for every disease in which there is any disturbance at all referable in any remote manner to a gland, we know of but a few mental conditions definitely of endocrinal causation. Myxoedema, cretinism or hypothyroidism, Graves' disease or hyperthyroidism, acromegaly (adenoma

very rare in children under the age of two, uncommon in the five years following puberty in both sexes, and only slightly prevalent in adult males under fifty years of age. On the other hand, it was enormously prevalent and severe in females from twenty to forty, somewhat less prevalent, but nearly always mild, in children of both sexes from two to ten years, and almost equally prevalent in old people of both sexes. The greatest pellagra morbidity was observed among persons engaged in housework or remaining at home without occupation, indicating that the causative agent or agencies are present in or near the home. Nevertheless, the women mill workers suffered as much as, or even more than, the housekeepers of the same age when due regard is given to the total number of persons thus engaged."

[23] When the situation concerning dementia praecox is considered I shall take up the endocrine theories that bear on its cause. Though there is a respectable amount of work to be cited in this disease no one as yet can claim any proof that the disease rests fundamentally in endocrine disorder.

A good deal of what passes as reasoning in the articles on endocrine disorder assumes that where endocrine disorder can be postulated it is necessarily *causative*. Conceding that in any given disease there are symptoms referable to the thyroid gland this may merely mean that the agent of injury *affects* the thyroid as well as the other parts of the organism.

of the pituitary), Addison's disease, the various types of infantilism, the mental difficulties of the menopause—these (except the last) play but a small numerical rôle in psychiatry. The mental disorders of the menopause are fairly frequent, and I shall present data concerning them in the section on family studies. Wherever heredity is invoked as the cause of the endocrine disorder the assumption of disease in relative or ancestor is so poorly grounded and so inadequately considered as to make it practically worthless. Whatever evidence we have concerning endocrinal mental disease, e.g., thyroid disease, is that it is essentially environmental in its origin,[24]

[24] I cite in this connection McCarrison (128) who shows that birds overfeeding, lack of exercise, and fecal contamination are potent in causing goitre. He is emphatic in pointing out the marked effect of diet either in a perverted form or in undernourishment on the vigor and vitality of the organism including the germplasm. He gives as the four great causes of endocrine derangement (1) direct disease of one gland or group of glands, (2) as a result of overstimulation due to acute infectious diseases, the exanthemata and bacterial toxins, (3) defective or improper feeding, (4) residence in unsanitary surroundings, intestinal toxaemia, etc. He points out that *if a healthy pregnant bitch has a large portion of the thyroid removed the pups are likely to be born with goitres.*

This is a remarkable experiment and one that bears heavily on the hereditary causation of disease. In other words, if a gland which has an important general function be injured the germplasm which has to do with the development of that gland in the unborn is involved, with the effect that the progeny are defective in more or less the same way as the mutilated parent.

Peru (128) gives facts about large areas of the world where goitre is very prevalent due entirely to the formation of the soil, the amount of calcium and the effect upon calcium metabolism. Tincture of iodine administered to all the living things in this community including the lowest animals as well as man produces healthy individuals. "There are large areas of the world where goitre is very prevalent—these are chiefly limestone districts and the relationship between goitre and such localities cannot be denied. The goitre found there is usually of the large type and if accompanied by any symptoms it is with those of hypothyroidism and cretinism. The only conclusions one can come to are that there are areas rich and poor in calcium so there must be areas rich and poor in iodine, and that it is possible to have a district rich in calcium and poor in iodine as well as one rich in iodine and poor in calcium. . . . . The balance between calcium and iodine content of the colloid has a very important bearing on the production of goitre."

It is interesting to note that those who know anything at all about it say little about heredity but have searched the environment for causes. Thus Crotti (37) concludes, after citing the literature, that while heredity is a factor in its etiology, that it can be accepted as an established fact that the goitre causative factor, whatever its nature may be is most frequently conveyed

i.e., results from infection, from food or drink deficiency, or arises spontaneously in hitherto "normal" families. This last is especially true of the cretins at the Waverly School for Feeble-minded.

to the organism through drinking water. After rejecting all other theories he accepts McCarrison's views, "So far the weight of evidence seems to be in favor of the infection theory."

But while stating heredity to be an important factor he cites nothing at all conclusive about this, whereas he cites classical instances (Bircher) where changing water supply relieved a whole population of endemic goitre. Add to this the work of Kimball, Rogoff, and Marine whose use of iodine as a prophylactic measure prevents goitre—and where is the *heredity* factor then?

CHAPTER IV

The Psychoneuroses, Neurasthenia, Psychasthenia and Hysteria

Though it would be logical at this point to continue with certain of the great psychiatric diseases, dementia praecox, manic-depressive insanity, involution melancholia, and senile dementia, it would not serve my purpose to consider them at this point. They form the basis of the family studies later discussed, and I shall therefore consider them just prior to the section dealing with the family studies. In this and the succeeding chapters I shall discuss three important psychiatric groups as to their value as hereditary factors. These three groups comprise the psychoneuroses, the epilepsies, and feeble-mindedness. I am considering these groups not because we are to study their heredity intensively, but because their occurrence in family groups is given a hereditary value. Thus if an individual has dementia praecox and one of his ancestors, collaterals or siblings, has epilepsy, or is feeble-minded, the two conditions are linked together as showing a common neuropathic heredity or background. I shall show that such a conclusion is by no means to be lightly reached, that there is no unity in nervousness, epilepsy and feeble-mindedness, that before any such condition occurring in the relatives of any person is given purely hereditary value it must be shown that it is not in itself due to environmental factors, is not the result of disease or injury, or some life condition.

THE PSYCHONEUROSES

So often it is stated in the histories of patients with mental disease that their father or mother, or some relative was "nervous," "very nervous," or suffered from "nervousness," that it becomes necessary to deal with this term and its implications both in disease and heredity. That non-medical investigators have been willing to accept the statement that an ancestor or a relative was nervous and to believe that this carried a hereditary neuropathic importance is not surprising. What is surprising is that medical men have been willing to so completely submerge clinical discrimination and judgment.

The fact is that the term nervousness as used by the ordinary

person in describing the case of anybody else or himself means anything from irritability to dementia, including fatigue, fear, restlessness, paralysis, criminality, fainting spells, headache, epileptic attacks, etc. The fact is that there is no disease "nervousness" and no unitary group of symptoms so to be classed. Further if the various things called nervousness constitute a neuropathic taint then none of us can escape, all of us have a neuropathic heredity, and the wonder is not that there is "inherited" insanity, but that there are any normal people. Indeed the same kind of reasoning which "proves" that "nervousness" of an ancestor is a background for mental disease in a descendant "proves" also that it causes normality.

When we come down to definite disease states we find that the problem connoted in the term "nervousness" is a very complex one and not to be settled by any off-hand summary. In the main there has arisen in the medical literature the term "psychoneurosis" to imply that the conditions classed under this heading are so-to-speak combinations of mental and "nervous" states (which frankly is a euphemistic disguise of the fact that in the main the psychoneuroses are really mental diseases). It is significant that the American Psychiatric Association has taken over these conditions as belonging to its domain rather than to neurology, and this is justified by the nature of the conditions.

There are three main divisions of the psychoneuroses; the neurasthenic, psychasthenic and hysteric. This division has been changed often enough—we hear of traumatic neuroses, anxiety states, etc.—just as we heard hypochondriasis and cerebrasthenia in the older divisions, but for the purposes of this book the division here used is satisfactory and embraces the bulk of cases. My problem is merely this, how far is the existence of these disease-states in the ancestors or in relatives of a patient with a mental disease to be given weight and importance? Because there are no statistics on the matter, no definite researches of value, no answer can be directly given and I must point out certain important relationships of these states as indirectly bearing on the problem.

NEURASTHENIA[1]

It is conceded by all practical medical men that a diagnosis of this condition is reached in great part by exclusion. This is tanta-

[1] For a discussion of the wide range of organic diseases in which neurasthenic symptoms arise see Mott (138). See especially Gerkie Cobb (27).

mount to saying that if after testing the patient in every way known to our science one finds no definite organic disease, *then and then only can the diagnosis of neurasthenia be justified.* In other words one may get the essential symptoms of neurasthenia, viz., the fatigue, sleeplessness, disturbance of digestion, pains and aches, mild depression, irritability and sex disturbance in tuberculosis, neurosyphilis, cardio-renal disease, cardiac disease, the various anaemias, etc. *Neurasthenia is a reaction, and occurs as a result of the lowered physical energy of disease or as a part of a mental disturbance arising from maladjustment.*

Thus Fales speaks of tropical neurasthenia as very common among Americans who go to the Phillipines, men and women suffering with it after they have resided there a year or more. Woodruff especially dilates upon this. Samson Hirsch speaks of the neurasthenia symptom complex observed in patients suffering from lead poisoning. "Neurasthenia," says Rennic, "must be carefully differentiated from myxoedema and Addison's disease." "All the symptomatic indications which had been relied on to establish the differential diagnosis of early general paralysis from ordinary neurasthenia are utterly fallaceious. . . . . The first positive signs of paretic dementia are in fact nothing but true neurasthenia."

I might multiply the above quotations to show that the neurasthenic state is a common reaction type to many diseases. If this is so, and if it takes a careful study of the patient at the hands of expert physicians to differentiate between the "true" neurasthenia and the types found as a result of organic disease, of how much value is the statement of a layman speaking perhaps of some one he has not seen for years or who perhaps died before he was born (that is about an ancestor or relative of a patient with mental disease) that he was "nervous"?

Yet it is this kind of evidence upon which "nervousness" as a background for "insanity" has its foundation. The evidence the law throws out as valueless—herasay and conjecture—medicine, in the field of psychiatry, accepts as truth.

It is true that neurasthenic states are often the equivalents of mild cases of such mental diseases as manic-depressive insanity, or involution psychosis, and the early stages of dementia praecox. It is often the case that the ancestor who had "neurasthenia" had these conditions but obviously this is a matter difficult to prove on the basis of a history.

Further, while neurasthenia is a reaction sometimes due to a morbid mental make-up, it is also the reaction of normal people to extraordinary situations. The War brought out a huge amount of neurasthenia in men in no way "tainted,"[2] energetic, able, and without the slightest previous evidence of a neurotic nature. It is true that it may be assumed that if they broke down it was because of a hereditary predisposition but this would be—and is—an especially common, and obnoxious case of begging the question. For example, "A" breaks down because he has an inherited weakness. But no one knew he had an inherited weakness, he seemed all right before this, and his parents and relatives are all well even if his mother has headaches occasionally. How does one know that he has an inherited weakness? "Because he broke down." Is not this vicious reasoning? Yet it occurs and recurs throughout the psychiatric literature. I am not here concerned with the question in how far neurasthenia itself is hereditary, though this would be a problem pertinent enough in a book dealing with the transmission of mental diseases. Most of the workers in the subject, even those who like Freud and Jung, trace its direct origin to psychological experiences of one type or another believe it has a constitutional basis. For example Allen says "Victims of neurasthenia are persons who in all cases have either inherited or acquired a nervous system with lessened power for active functioning. There probably is in neurasthenia an imparied metabolic power. The nerve cells break down too easily and build up too slowly." Certainly there is not the slightest evidence, clinical or pathological, for the last statement, just as there is no evidence that the neurasthenic has "either inherited or acquired" (how else could he get it) a nervous system with lessened power of functioning. In fact the ablest people artistically and intellectually, are more liable to neurasthenia than the feeble-minded, and city people are more liable than country people. Does anyone suppose that the artistic and intellectual have less able nervous systems than the feeble-minded or that city people are mentally deficient as compared with country folk?

Sajous (117) says,

> Heredity acts only as a predisposing influence, through parental neuroses or psychoses. (Then) Gout, rheumatism, syphilis and tuberculosis may act

[2] See Southard's (181), Shell Shock.

as predisposition conditions in the offspring. Excesses of all kinds, particularly in sexual relations, lower the resistance of the organism as a living entity (not only of the nervous system) and pathogenic factors find a fruitful field, which had not inherited depravity prevailed would have proved sterile.

A delightful statement! Few are the fish that can escape the net of "inherited depravity" (whatever that may be). (1) Parental neuroses and psychoses, including a dozen conditions to which a neurosis or psychosis is incidental, (2) Gout, rheumatism (a term given to a half dozen and more conditions of widely differing origin), syphilis and tuberculosis, these last two perhaps the most common of conditions. (The Jews have had less syphilis and tuberculosis than any of the great white races—but no one accuses them of being *less* neurasthenic than other races.) (3) Excesses of all kinds—including venery, eating, drinking, working, and who knows, thinking? The wonder is that any have escaped neurasthenia.

My own opinion is that since Morel's time and Beard's day too few have critically examined the data of neuropathic heredity, that many of the writers and workers have been hypnotized by the beautiful generality popularized by Morel (136) and Esquirol, and pushed by Beard (10), that all forms of disease are interchangeable and that heredity means anything pathologic or queer in ancestors or relatives. Buckle's words ought to be ingraved in every clinic and on every writer's desk. My belief is that *some* people *are* predisposed to neurasthenia, in that from the first days of their lives they showed lowered energy and endurance, irritability, emotional unrest, etc., but that *any* man or woman *may* become neurasthenic regardless of heredity, race or physical make-up, that in some cases the main element seems to be in the individual, that in vastly more cases the disturbing element is mainly the environment. When one sees one neurasthenic in a large family of apparently normal people, and when one sees masses of hitherto "normal" men breaking down under war conditions, when one sees sturdy ancestors, stolid immigrants, giving issue to restless, sensitive, nervous "Americans"—it becomes difficult to believe that a true heredity has much to do with the most of neurasthenia.

The point is that to include neurasthenia as a hereditary cause of mental disease in a descendant is to beg a dozen questions, not merely

one, for in any history where neurasthenic-like symptoms are described one can only guess their real nature.

PSYCHASTHENIA

What has been said of neurasthenia is true, but in lesser degree, of psychasthenia. Psychasthenia is more distinctly a mental disease than neurasthenia, since its main symptoms are "morbid fears, imperative ideas, doubting mania and morbid impulses" (Allen). There is no sharp line between psychasthenia and hysteria and indeed it is now the custom to class as anxiety hysteria the fear state of psychasthenia (Freud, Finck, American Psychiatric Association classification). The hypochondriac states into which the psychasthenic falls have a close resemblance to the somatic delusions seen in involution diseases, manic-depressive insanity, and dementia praecox, and there is no doubt that psychasthenia often reaches the point where commitment to a hospital for the insane is necessary. In this it differs from ordinary cases of neurasthenia. Moreover, the psychasthenic beginning to dementia praecox and the other diseases is very common, and it is my belief, which it is difficult to prove, that in many, perhaps most cases, psychasthenia really represents abortive cases of these diseases. The anhedonic syndrome which I have elsewhere described[3] is an essential symptom complex in much of psychasthenia and also enters prominently into the psychoses. I am therefore prepared to believe, in view of what I shall later show, e.g., that a case of hypochondriacal psychasthenia in an ancestor may well be related to a case of manic depressive insanity in a descendant, but there is no way of proving this at present.

HYSTERIA

"Hysteria is a morbid state produced by 'suggestion' and removed by 'suggestion'" (Babinski). "Hysteria in its broadest sense is not a specific disease but a morbid biological reaction in which morbid bodily disturbances are caused by more or less subconscious mental states" (Allen).

[3] Myerson, A. (143), Anhedonia.

This remarkable condition was known from the earliest times, and thus is in marked contrast to neurasthenia which came into prominence with the writings of George M. Beard, the American neurologist. It is marked, whatever its cause, by an enormous variety of symptoms, ranging from the hysterics, "fits" of laughing and crying, to hysterical convulsions, including the remarkable losses of motor power, the paralyses, the losses of sensation, the anaesthesiae, and such symptoms as amnesia, flights, confused delirious states, vomiting, etc. The hysteric notoriously uses the symptoms for one purpose or another, to win a domestic battle, to triumph in litigation, to be excused or set free from a disagreeable situation, to win sympathy—in a word hysteria is the weapon of a weakness which refuses to accept defeat.

Whatever the theories that explain hysteria they all rest in the last analysis upon a congenital weakness. Even Freud, with his complicated psychoanalytic procedure, though it lead through a maze of repressions, conversions, complexes, censors, subconscious strivings and incest motives, brings one to a foetal state. Janet, with the lowered psychic tension as his starting point, is careful to tell the reader that his patient's parents were heavily alcoholic and a brother insane. The constitutional psychopathic inferiority of the hysteric is stressed in the literature and Moebius links it with neurasthenia as the mother stuff of insanity.

My own studies of the ancestry of the inhabitants of the insane hospital does not show any great occurrence of hysteria. In fact, it is never specifically mentioned, but this may be because it is masked under the caption "nervousness." Nor in reading the case histories of other workers, later to be cited, do I find that hysteria is disproportionately mentioned or implied in the ancestry of the insane. One who sees a severe hysteric realizes that the patient acts "crazy" but in so far as I can see this disease does not beget dementia praecox, manic-depressive insanity or the like. It may be that the hysteric patient comes of a "nervous" family, and indeed he or she often does, but as often does the patient spring from a family who wonder at him as an anomaly, some one not to be explained by heredity. Nor in my own practice have there been many instances where several members of the family had hysteria, which experience is quite different from that seen in dementia praecox or manic-depressive insanity.

The war brought an immense amount of hysteria in men accounted normal. The impression that the army neuropsychiatrists brought away from their experiences is that hysteria is a reaction type, a psychological dissociation brought about by situations to which adjustment is difficult and from which severe inner conflict and emotion arises. The war demonstrated that hysteria is induced in the male by trauma and fear, and that fine typest of men may be victims as well as those to be classed as psychopathic inferiors.[4]

I do not doubt that much of hysteria is congenital or constitutional. But we must sharply discriminate between congenital, constitutional, and hereditary. If for the time we leave out of consideration the proposition that the germplasm of the parent may be injured, there is no serious doubt that the developing embryo, or the very young infant, may be altered by disease. Those who have seen adults altered in character and temperament by a passing illness or injury will not be surprised or unwilling to believe that the destiny of a life may be altered by nutritional disturbance, injury or even early bad habit formation. *In other words, while it has been recognized that "congenital" does not equal "hereditary," it has not been emphasized that "constitutional" may arise in the germplasm but also at almost any time from conception to death, i.e., constitutions may be altered in utero, at birth, during childhood and even in adult life by infection, trauma and the wear and tear of existence.*

SUMMARY

There are cases of neurasthenia, psychasthenia and hysteria which stand in some familial relationship to other mental diseases. This is because there are cases of these conditions which are essentially minor cases of dementia praecox, manic-depressive insanity or involutional disease. Many a "nervous breakdown" in puberty, maturity or the menopause is really a psychosis as the term is ordinarily used, and such a "nervous breakdown" in an ancestor may stand as equivalent to the more serious mental state of the descendant. But the most of neurasthenia or of hysteria (and possibly what is to follow is true of some psychasthenia) has no such relationship, has a huge variety of causes, represents reactions of the human being to

[4] Southard's (181), Shell Shock.

physical illness and difficult environmental situations, is not unitary and to be classified in a simple manner. When "nervousness" is stated to have existed in an ancestor or a relative and it is then naïvely concluded that the "nervousness" has a hereditary importance, the conclusion violates all the laws of logical thinking, assumes what is to be proved, and throws overboard the kind of knowledge called clinical.

CHAPTER V

Epilepsy

Among the outstanding diseases of mankind epilepsy occupies a place of its own. Like its distant relative, hysteria, it has undoubtedly played its part in the organization of religion and the destinies of nations. It is well known that the "sacred disease" as the ancients knew it, helped give rise by its manifestations to the ghost-soul belief and to that theory of demoniac possession which still lingers in regard to mental diseases.

Our present point of view regarding epilepsy may be briefly stated, (1) we no longer hold it to be a unitary disease, but speak of it as divided into several syndromes which are grand mal, or the major classical seizures; petit mal, the momentary periodic loss of consciousness without the "fit"; Jacksonian epilepsy, or the spasm in a local part, not generalized at least at first, and psychic epilepsy, or that queer substitute for the seizure in which the patient, unconscious, carries out actions in a violent hurricane-like manner. (2) As causes of "the epilepsies" we recognize organic conditions, such as neurosyphilis,[1] brain tumor, arterial disease of the brain, multiple sclerosis, etc., toxic conditions such as alcoholism, plumbism, and the toxic states of pregnancy, "reflex" causes such as gastro-intestinal stasis, bad diet, ear and nose conditions, and finally there remains a large group of cases in which we find no known cause and thereupon beg the question by calling such cases idiopathic, implying that it is constitutional and innate. Every now and then some one "discovers" a bacillus which causes the disease, or else that the large intestines are responsible. At the present we had better disown the term idiopathic epilepsy, search diligently in each case for a cause, and not finding any say that the cause for this case is unknown.

This is a simple statement of the status of epilepsy. How does it compare with the expression of the older authorities and older

[1] It is very interesting to note the observation made by the Solomons (178). The Wassermann positive of adult epileptics gives less than 2 per cent positive, which is no more than the average community. When youthful epileptics are considered somewhere around 20 per cent gave positive Wassermanns.

principal writers on this subject? They are almost unanimous in placing the main burden as cause of epilepsy upon—heredity. I shall, in the following pages, cite them and their opinions, and state my objections to their conclusions.[2]

Oppenheim (148) writes:

> According to the unanimous experience of writers on the subject such as Herpin, Moreau, de Tours, Berger, Neckas, Echeverria, Gowers, Lange, *heredity* is the most important on one of the most important of the many causes of the disease. A number of conditions are capable of producing it but apparently they make their influence felt chiefly when the morbid predisposition is already present. A hereditary tendency to disease (??) may be ascertained in about one-third to one-half of the cases, in 35 to 40 per cent according to Benswanger, and in nearly three-fourths according to Finht. In almost two-thirds of these cases the heredity is direct.

Criticism. First, the authors cited are most of them old authors, who believed firmly that insanity was a unit, did not know the origin of general paresis in syphilis, believed that tuberculosis was inherited, made much of the scrofulous constitution, spoke of alcoholism as a neuropathic character, and in no case had any control figures as to the ancestry and relatives of the normal non-epileptic population. Further, most of them did not separate the various types of epilepsy one from the other. The work of Echeverria, Gowers, and Binswanger I shall criticize on the basis of their own writings.

[2] It is fair to say that some of the older authors did not agree with the opinion that epilepsy is hereditary. Thus Morel says it is not transmitted from parent to child, and Lasèque "considers epilepsy as an acquired infirmity, and resulting either from traumatisms producing permanent lesions or from spontaneous malformations" (quoted from Ch. Féré (53), La Famille Neuropathique). Féré is a strong adherent of the neuropathic relationship of epilepsy (see Féré for a full citation of many older authors). Féré by inference pulls into epilepsy, as part of it or related to it, migraine which is a "partial sensorial epilepsy," spasm of the glottis, asthma, Menière's disease, eclampsia, incontinence of urine, etc. Féré has the convenient assumption that if a "fever" or an injury start epilepsy they do so merely by releasing a predisposition. Applying this kind of reasoning Spirocheta Pallida does not cause syphilis, it releases a predisposition to syphilis, meningococcus does not cause meningitis, but releases a predisposition, etc. Medicine cannot yet afford to indulge in such subtleties. Féré, like many of the older authors, stresses the alcoholism of the parent, and especially drunkenness at the time of conception (Esquirol, Seguin, Morel, Lucas, etc.).

Second. "A number of conditions," says Oppenheim, "are capable of producing it (epilepsy) but apparently they make their influence felt chiefly when the morbid predisposition is already present." Why invoke predisposition when a condition can produce epilepsy by itself? Oppenheim, brilliant clinician that he was, mentions as causing epilepsy acute infective disease, syphilis, injury to head, heart disease, disturbances of circulation due to athermo, reflex causes and fright. If a fractured skull can produce epilepsy in any one, providing the fracture and all other resulting circumstances (hemorrhage, etc.) are adequately arranged, why speak of predisposition? If a child has an infectious disease with a resultant encephalitis and has epilepsy, why invoke predisposition? Any one may get epilepsy if a lesion be placed properly.

Third. "In about two-thirds of these cases the heredity is direct." No one else makes this statement. In fact the weak point in establishing the heredity of epilepsy is the rather *infrequent* occurrence of epilepsy in relatives. (See Gowers (69), Spratling and Thom (195, 196).) Oppenheim mentions without criticism the fact that the great neurologists Marie, Freud, and Allen Starr found in postmortem examinations that all epilepsy was of organic nature, i.e., was caused by definite lesions.

Says Gowers,

> There are few diseases in the production of which inheritance has more manifest influence and traceable influence despite the fact that traceable influence is always far less than that which exists. In examining inherited influence it is necessary to inquire: (1) How is its existence shown? (2) In what proportion of cases can it be traced?

As a result of his own researches he believes that "insanity" is the simple largest hereditary factor, "insanity" in parents, near relatives (uncles, aunts), brothers and sisters. My objection to such a conclusion is (1) it is contained in the premise —viz., insanity in an ancestor or relative is defined as a hereditary factor in the production of epilepsy in a descendant or relative, but there is really nothing to prove this except possible percentages, (2) the term insanity may mean senile dementia, general paresis, dementia praecox, etc., and does not necessarily have any familial bearing (see first chapter), (3) what normal controls has Gowers?

Gowers hits heavy blows at some favorite inherited taints. "Intemperance is probably also due in many cases to a neuropathic

disposition, but is seldom to be trusted as to its (hereditary) evidence." He further states that tuberculosis is not at all to be trusted in such a relation. Even hysteria is "too closely related to female temperament" to be a neuropathic taint. "Migraine is too closely related to hereditary gout to permit weight to be assigned to it." I cite these conclusions in relation to the statements of Davenport and others, considered later.[3]

Spratling. He quotes Féré approvingly, "As a matter of fact we must look upon epilepsy as a group of symptoms which may appear in greater or lesser number in the course of very different pathological conditions, sometimes under one form, sometimes under another. . . . . Epilepsy applies solely as a symptom complex."

Then, after so accurately sizing up the situation, he goes on to speak of epilepsy as a *disease* dependent on nervous or degenerative disease in the parents more frequently than in any other cause! He says that 16 per cent of cases show similar heredity, and cites just one case, in which his information as to the condition of the child comes from another physician! His dissimilar heredity statistics are based on the assumption that insanity, nervousness, migraine, chorea, etc., in relatives is a hereditary cause of epilepsy in any individual. I have criticized this so frequently that I forbear doing so in any detail now.

Binswanger (14). For a thorough account of the literature concerning the heredity of epilepsy Binswanger is to be praised. In this book he gives the figures of Moreau, Echeverria, Gowers, Dejerine and many others in relation to this question. All of these workers agree as to the hereditary nature of epilepsy and Binswanger himself gives an account of his own investigations. Since the methods of these authorities are the same I shall cite Binswanger's own work.

Binswanger is a believer in the theory laid down by Morel and Esquirol, that the forms of mental and nervous disease constitute a diverse manifestation of a unity, a neuropathic or psychopathic predisposition which may be (1) inherited, (2) developed intra-uterine, (3) developed in extra-uterine life. The first two are classed as congenital neuropathic constitution (Veranlangung), the inherited

[3] He also quotes, with complete accord, Herpin, who states that hemiplegia and allied forms of paralysis occur less frequently in the parents of epileptics than in the parents of the normal population.

constitution is especially designated as neuropathic taint. The third type is to be called acquired neuropathic condition.

1. The neuropathic taint in Binswanger's usage is not necessarily a heredity, in the Weissman sense. It may arise through injury of the germplasm. The most common sources of this germplasm injury are choreic, intoxicative (alcohol, morphine, lead, etc.), infectious (tuberculosis, syphilis), constitutional diseases (anemia, leukemia, chloritic diseases, diabetes, gout, chronic hypertrophic arthritis), and finally local diseases of the germ-forming organs, testicle and ovary through mechanical injuries, or degenerative or inflammatory processes.

It is important to emphasize that Binswanger is not in reality discussing the heredity that Davenport, e.g., discusses. He is discussing germplasm injury. He is declaring that the environment through some infection or toxic process may injure the germplasm so that it transmits damaged individuals. This would imply that were these toxic influences—syphilis, tuberculosis, gout, and alcoholism—removed from the world better individuals would result, which is contrary to the belief of most of the biological writers on mental disease. Their implication is, as I shall show later, that these toxic influences serve to eliminate the weaker individuals and serve to cleanse or purify the race as fire cleanses or purifies metal of dross. Following Binswanger's idea of germplasm injury to its logical conclusion, and he lines himself up in opposition to the ideas of the eugenists. As will be evident later, I thoroughly agree with Binswanger in this attitude, namely, that toxic agents can and do injure germplasm and though they may or may not be behind epilepsy and mental diseases they are productive of harm to the race as well as to the individual, and that logically we must look for such causes of degeneracy rather than to the abstract conception heredity.

When Binswanger's table on page 62 is closely examined certain facts stand out. Alcoholism is present in 51 per cent of the cases in some ancestors. As before noted Gowers does not believe that alcoholism plays a rôle in epilepsy, and we are not informed by Binswanger just what he calls alcoholism and how large a percentage of the population from which his epileptics come are alcoholics by his definition. Are epileptics less common in non-drinking peoples, say the Jews? This question would be hard to answer, but it is my impression that epilepsy is fully as common among them as among

Disease table of hereditary taints on maternal and paternal sides.—Binswanger

DISEASES	MATERNAL SIDE			PATERNAL SIDE			OTHER RELATIVES	TOTAL
	Mother	Grandfather	Grandmother	Father	Grandfather	Grandmother		
Alcohol	7 = 2.8%	14 = 5.7%	2 = 0.8%	92 = 37.7%	16 = 6.5%	4 = 1.6%	1 = 0.4%	136 = 51.6%
Migraine	44 = 18.1%	1 = 0.4%	2 = 0.8%	9 = 3.6%		1 = 0.4%	3 = 1.2%	60 = 24.5%
Epilepsy	6 = 2.4%	2 = 0.8%		3 = 1.2%		2 = 0.8%	39 = 15.9%	53 = 21.2%
Insanity	2 = 0.8%	1 = 0.4%	2 = 0.8%	11 = 4.5%	1 = 0.4%	4 = 1.6%	20 = 8.1%	41 = 16.8%
Hysteria and hysteric epilepsy	18 = 7.3%		2 = 0.8%			2 = 0.8%	6 = 2.4%	28 = 11.3%
Nervousness	16 = 6.5%			1 = 0.4%	1 = 0.4%			18 = 7.3%
Suicide and suicidal attempts	2 = 0.8%	2 = 0.8%		8 = 3.2%			6 = 2.4%	18 = 7.3%
Convulsions							15 = 6.1%	15 = 6.1%
Idiocy							6 = 2.4%	6 = 2.9%
Imbecility	1 = 0.4%						3 = 1.2%	4 = 1.6%
Neuralgia	4 = 1.6%							4 = 1.6%
General paralysis	1 = 0.4%		1 = 0.4%					2 = 0.8%
Strabismus	1 = 0.4%						1 = 0.4%	2 = 0.8%
Deafness				1 = 0.4%			1 = 0.4%	2 = 0.8%
Blindness				1 = 0.4%				1 = 0.4%
Clubfoot	1 = 0.4%							1 = 0.4%
Facial tic	1 = 0.4%							1 = 0.4%
Hemiplegia						1 = 0.4%		1 = 0.4%
Ataxia		1 = 0.4%						1 = 0.4%

the drinking populations. However, if alcohol does cause epilepsy then we are not dealing with fortuitous heredity, we are dealing with germplasm injury, a quite different matter, and a difference of fundamental importance.

In 24 per cent of the cases *some* relative had migraine, and in two-thirds of the cases this was the mother. This is extraordinary, and I wonder, just a bit, if Binswanger's investigation did not include headache in his statistics. Headaches are very common in women. We are not told just how the diagnosis of migraine was reached. There are those who believe that migraine is a form of epilepsy and they would receive some support for their views from Binswanger. But the cause of migraine is really unknown and most writers incline to the belief that some auto-intoxication is at work. According to these figures migraine of the mother is an important factor in relation to the epilepsy of a child. I may say that Binswanger's experience is far different from mine. In the last 100 cases of epilepsy which I have examined I have specifically inquired for migraine, and have found it 3 times. Headache was common, but headache is not migraine. The best way of studying this question would be to study the descendants of *known* cases of migraine. I venture the prediction that epilepsy would be as common but no more so than in the population as a whole.

Epilepsy was found in the direct ancestors in a total of 10 cases out of 250, or about 4 per cent. If this includes all forms of epilepsy and convulsions, it is not a large percentage. There were 39 cases of epilepsy in all "other relatives," which can only have a very indirect bearing since the germplasm, let us say of a cousin, is in only a fraction similar, and the epilepsy of a cousin may have an entirely different value because of a different origin. But most certainly even Binswanger would hardly agree that Echeverria's 51 per cent insane ancestry was justifiable.

Insanity occurred in 21 direct relatives or in about 9 per cent. Since we are not told what form the insanity took, this information is of little value. Mental diseases are very common, and are more common wherever life is prolonged well into old age. If senile dementia of one form or another is included 9 per cent is not very high in parents and grandparents.

I shall not discuss the other headings as I have voiced my objections to them repeatedly. That there was only 1 imbecile in the

direct ancestors of 250 epileptics seems difficult to believe. It would be hard to find 250 normal persons with as good a record. Further, there is apparently a protective effect in having epilepsy, for it seems to shield ancestors from hemiplegia. In this series it occurred only once, or much less often than one would find it in the history of any normal group of 250 people. Similarly with general paralysis, strabismus, blindness, deafness, club foot, facial tic and ataxia.

Davenport (40, 41). One of the latest of those to express ideas on the heredity of epilepsy is Davenport, the director of the Eugenics Laboratory at Cold Springs Harbor, New York, an eminent biologist, whose attention to eugenics has received world wide attention. In much of his work he has had medical collaboration, but except in a few instances he has directed the work of the medical men along the lines of this thought. Davenport is a Mendelian and I am frank to say that he seems to me determined to find in the mental diseases a Mendelian significance and postulates in advance conclusions which he is able to verify. I shall cite his work frequently and must therefore give some attention here to his general method.

Patients are selected, usually from an institution, and then their ancestry and relatives are looked up by a social worker, usually a woman with a very limited training in medicine, who goes into the community interviewing relatives, neighbors, friends, social agencies and studying records. This information is the chief basis for the conclusions reached, and to a medical man the sang-froid with which the social worker makes diagnoses on people she has never seen, or else met in a casual way, is nothing short of appalling. Really, it seems utterly unnecessary to have laboratories, blood tests, psychological tests, clinical examinations, and to take four years in a medical school plus hospital experience, etc., when a woman can as a result of a dozen or two of lectures make all kinds of medical, surgical and psychiatric diagnoses in an interview or by reading through a court record.

I introduce at this point the legend which appears frequently in the article here discussed, and in one form or another, in most of Davenport's work on psychiatric problems. I have added to it, in parentheses against each trait or class of mental condition a few remarks of my own, which of course indicate my quite hostile opinion.

"Frequency of the different classes of mental conditions in the children of two mentally defective parents together with the mental

condition of the grandparents, the parent's siblings (brothers and sisters), and other blood relatives about whom something is known."

I need not say that Davenport is not essentially responsible for this legend and in reality my rather harsh criticism is directed more against his medical collaborators than against him.

a, alcoholic (See chapter III, p. 33, etc. Also Gowers' opinion, p. 59)
ap, apoplexy (More common in relatives of normal. Not primarily a psychiatric problem, a circulatory problem)
b, blind (Most common cause, gonorrhea. Other causes syphilis, trauma, cataract, ocular infection, none of which is in any way "mental")
bd, affected with Bright's disease (A disease or disease-group of the kidney)
c, criminalistic (There are all kinds of criminals as everybody knows; social criminals made such through environment, and accidental criminals made such by some group of circumstances operating, and undoubtedly a large number of psychopathic criminals from "feeble-mindedness," "insanity." There is no unit to criminality as all criminologists know, and it is absurd to speak of criminalistic as a psychopathic trait. Further progress in criminology depends on study of "the *individual delinquent.*" Vide Healey)
ca, cancerous (The problem of cancer is not a problem of mental disease in the least degree, nor is it even definitely understood to be hereditary)
ch, chorea (As time goes on Sydenham's chorea, which is the most common form, is definitely understood to be an infection, usually arising from the tonsils or some other source, and frequently associated with endocarditis)
cr, cripple (Accident? Injury? War? Old person through rheumatism? Young person through a congenital disease? One could make a list of cripples a page long in whom there was no question of mental disease)
d, deaf (The causes of deafness are so many and in the main so related to environmental disease that it would be humorous if it were not pathetic to have it included in mental traits)
dfm, deformed (See cripple)
dp, dementia praecox (Certainly a psychopathic trait but how is the conclusion reached, hospital records or the happy guess of the field worker?)
dt, delirium tremens (Diagnosis made how? For further remarks see page 33)
dw, dwarf (Means nothing psychopathologically. Dwarfs are often insane or feeble-minded, but every dwarf is not)
e, epileptic (Again, how is the diagnosis reached?)
ec, eccentric (My barber thinks I am eccentric because I read part of a book whenever I am waiting for him. Certain eccentricities are psychopathological, others are not)
en, encephalitis (A problem of infection having no psychopathic taint. There was no selection of psychopathic types in the last few epidemics)

f, feeble-minded (See later chapter on feeble mindedness. Question, how is the diagnosis reached?)
go, goitre (See page 45. It is well understood today that most of goitre is the result of environmental conditions. The problem of exophthalmic goitre is certainly not solved, and no one has a right to speak of it as a psychopathic taint as yet)
gp, general paralysis of the insane (See page 27–30)
hd, heart disease (This is as much a psychopathic taint as bunions)
hy, hysteria (See page 53. Also note Gowers' remark on the same, page 60)
i, insane (This whole book is directed against the use of this term as carrying a unitary meaning)
id, ill-defined organic disease (Worse than ill-defined as a psychopathic trait or a mental disease)
kd, kidney disease (See bd—Bright's disease)
la, locomoter ataxia (The disease, tabes dorsalis, for which this is a synonym, is syphilitic and has the significance of syphilis only)
sb, softening of the brain (I do not know what that means)
sc, scoliosis (Twists in the brain do not follow twists in the spine)
sd, senile dementia (See page 97)
sh, shiftless (May or may not be due to mental disease)
sm, simple meningitis (I have never heard the diagnosis before. Certainly has nothing to do with heredity)
st, stillborn (? ? ?)
su, suicide (All the writers on suicide have emphasized the fact that suicide is a queer act, but there are many types of people who commit suicide. Some are psychopathic, some have mental disease, others are normal individuals reacting to difficult situations)
sx, unchaste (If that is a mental disease Heaven help society!)
t, tuberculosis (Tuberculosis is an infection arising from environmental conditions and not running parallel to mental disease)
tf, typhoid fever (I am not sure that this is to be seriously considered. See remarks on cancer)
va, varicose veins. (See remarks on typhoid fever)
ve, vertigo (Vertigo is a symptom arising in a dozen or more different diseases, and has absolutely no relationship to mental disease and especially none to heredity)
w, vagrant (See remarks on shiftless)

In many other places in this paper Davenport states that there is some evidence of mental weakness implied by the terms migrainous, choreic, neurotic, and paralytic. I deny this implication completely. *Paralytic* has absolutely no implications of mental weakness, neither has neurotic. *Choreic* may possibly have a neurotic component but the trend of opinion is directed distinctly towards its being an infection. As to *migraine* its nature is as yet unknown. He

enumerates ten main classes that are tainted—epileptic, feeble-minded, insane (all of which are very objectionable as class terms) migrainous—which I have already criticized, neurotic which I have discussed under the term psychoneuroses, alcoholic—which may or may not be "tainted" in origin, paralytic, sex-offender—this certainly has no implication of mental weakness else a large majority of the men of the community would fall under this heading, and some of the finest types mentally in all history would be stigmatized. There are sex-offenders who show mental difficulty just as there are biologists, neurologists and college professors who do. The term itself and the act or acts have no such implication. Choreic, suicidal and criminal—these I have already sufficiently criticized.

Needless to say that I do not feel that Davenport is warranted in his conclusions regarding epilepsy which are that "no epileptic transmits like a normal," "that the normal parent of an epileptic usually has some defective germ cells" (even, I suppose, if the child fractures the skull and develops epilepsy), and that epilepsy is a hereditary Mendelian character due to a defect in the germplasm. As the Mendelian hypothesis will appear later as a leading explanation. I shall defer further consideration of this phase of Davenport's until then. Meanwhile, it seems to me that Davenport and his followers have been dogmatic offenders against logic and science —they have collected data in a thoroughly unscientific way, they have unified utterly diverse conditions into one "neuropathic defect due to lack of a unit determiner" which last is an arbitrary conclusion decided upon, apparently, beforehand.[4]

It is interesting to note that in his latest writings on epilepsy, Davenport has greatly modified his opinion. In studying the incidence of epilepsy, he finds it is widespread among the higher vertebrates. He now says, *an* if not *the* hereditary factor behaves like a recessive. He winds up as follows: in general the statistics suggest that alcohol may be an important exogenous factor which influences the epileptic attack, but doubtless the constitutional factors are no less important.

[4] Whoever thinks that the above statement is too severe and polemic is referred to the article by David Heron (77), "Mendelism and the problem of mental defect: A criticism of recent American work." I have been very mild in my statements compared to those of this writer and that Karl Pearson, in two other articles entitled "On graduated continuity of mental defect." On page 280 of this volume, I give some of the statements of these writers.

Thom (195, 196). Douglas A. Thom has carried on a series of investigations bearing on the question, not of the relation of epilepsy to other mental diseases, but on the fate of the offspring of epileptics. At this place it is again necessary to point out that there are two main divisions of epilepsy, one in which the epilepsy is held to be primary, that is without known cause, the so-called idiopathic epilepsy, and a smaller group in which the epilepsy is held to be organic in origin, that is results from definite brain disease. It is worth while to state that such workers as Pierre, Marie, Freud, and Allen Starr believe on the basis of post-mortem work that all epilepsy is organic. To speak of a symptom complex such as epilepsy as primary or idiopathic seems as reasonable as to speak of the symptom complex of "cough" as idiopathic.

The most interesting of comparisons is to be found in the relative figures of Echeverria and Thom on the frequency of epilepsy in the offspring of known epileptics. In his second paper Thom makes a table of comparison between his results and those of his predecessor.

INVESTIGATOR	MARRIED EPILEPTICS	MALES	FEMALES	NUMBER OF CHILDREN	DIED IN EARLY INFANCY OF CONVULSIONS	EPILEPTICS
Echeverria	136	62	74	533	195	78
Thom	138	68	70	553	6	4

"That 273 offspring out of a total of 533 or over 51 per cent should have developed epilepsy seemed more startling to me than my own figures of only 10 epileptics in my total of 553 offspring or less than 2 per cent" says Thom.

It seems to me that Thom writing in 1920 is more to be credited with belief than Echeverria writing in 1884. Since the latter's time medicine has made great strides, and there are exacter and better means of obtaining information. The people Thom had to deal with lived in a community where records, transportation, etc., are better and more available.

Further, death in early childhood from convulsions is not necessarily epilepsy as both Echeverria and Thom seem to assume although what is called convulsions is later followed by epilepsy.[5] It is a

[5] Patrick and Levy (152), in a study of early convulsions in epileptics and others, find that there were nearly five times as great an incidence of early

common cause of death and arises from many causes, such as bad diet, infectious diseases of childhood, injury, etc.

I cite Thom's conclusions in full.

1. The records of 1536 epileptic patients admitted to the Monson State Hospital showed that 1 out of every 6 was married.

2. Eighty per cent of the marriages resulted in children.

3. One hundred and thirty-eight marriages resulted in 553 offspring, an average of 4 to each marriage.

4. Of the 553 offspring it was possible to obtain a history of epilepsy in only 10 cases (1.8 per cent).

5. Of these 10 epileptics, 6 died in infancy, 2 became confirmed epileptics, and 2 cases are arrested, one for seventeen years, the other for thirty-six years.

6. The study of the family history in 138 epileptics revealed epilepsy in one parent, 8 times, and the study of the offspring of these same patients showed that it had been transmitted 10 times; but in 2 instances there were 2 children in the same family who had convulsions, so that the epilepsy was inherited and transmitted in 5.8 per cent of the cases.

7. These figures correspond more closely with the recent work of Stuchlik, who found inheritance of epilepsy in epileptics direct from parent in 4.1 per cent of 176 cases, than they do with the findings of Turner, Doran and Binswanger.

8. From the above it would appear that epilepsy is transmitted directly from parent to offspring less frequently than we have heretofore been led to believe.

Thom contrasts the offspring of patients with "idiopathic" epilepsy with the offspring of patients with organic epilepsy. The essential defect in this table is that the children of no group of normals are used as a control. If it were I predict freely that there would be nearly as many of the children of "normals" having convulsions as the children of epileptics.

It will be seen that Thom is guardedly optimistic in relation to the heredity of epilepsy. His work is to be criticized in that he too has accepted epilepsy as a disease in his conclusions (though he specifically states it is not elsewhere), and that when he says "it is the nervous system" lacking in nervous stability that is in-

convulsions in the epileptic group (500 cases) as against the unselected group (752 cases). The authors conclude that there would be a larger percentage of convulsions in the history of epileptics if it were not for faulty memory. Multiple convulsions occur much more frequently in the epileptics than in the non-epileptics. The non-epileptic convulsions are apt to be brief, generalized, and not followed by confusion.

herited and the manifestations of this instability may be mental deficiency of all degrees, insanity of various types, neurological and psychopathic disorders, convulsions from various exciting causes, he makes the same error that I have so freely and perhaps presumptuously criticized in the other writers. He takes for granted what he has to prove, and in fact what is now known not to be true. It may be that an epileptic has an extraordinary reactivity to stimuli from the gastro-intestinal tract, from eye-strain or from emotion; it may be as L. Pierce Clark (25) states that there is a characteristic epileptic temperament, but even if this be true it may be *constitutional* without being *inherited*. Thus it may be the result of intrauterine disease or of early injury without being a hereditary character. It is one of the trite observations in neurology that a head injury alters character in a remarkable way, brings about irritability, egoism and even epilepsy, and it is not at all unlikely that injury of this type from the earliest days of childhood may have a far reaching effect in relation to epilepsy.[6]

There is a curious fact which I have unearthed which bears heavily on the whole problem. If epilepsy is a hereditary disease one would expect it to occur frequently in siblings, i.e., brothers and sisters. No matter what its origin in a preceding generation, "nervousness," "insanity," "criminality," it ought to appear in a similar form in members of the same generation, for this is the rule in mental diseases and feeble-mindedness. What are the facts? I contrast them with those found in feeble-mindedness and "insanity."

1. At the Waverly School for the Feeble-minded there are at the present time 60 families of brothers and sisters, numbering 130 individuals in all, this out of a total population of 1500. At the Wrentham School for the Feeble-minded there are at present 80 families of brothers and sisters, numbering 300 individuals, out of a total population 1300! (I do not count cousin families, nor families of which one member is now present out of several who have been pres-

[6] I have omitted discussing Brown Sequard's work on the production of epilepsy by traumatizing the sciatic nerve of guinea-pigs and the transmission of the condition to the descendants. Though this work was in part confirmed by Romanes, later writers have failed to corroborate it. It is true that epilepsy can be caused in this way, but it is very doubtful whether this form of epilepsy is hereditary. See the criticism of Wizosch-Maidsza in the Archives f. Rassen und Gesellsch. Biologie, 1914, 11, page 289.

ent.) Thus there certainly appears to be a factor either hereditary in nature or injuring the germplasm of the ancestors, in the case of feeble-mindedness.

2. In the Taunton State Hospital, which I have especially studied, there were in 1917 about 160 related persons, mostly brother and sister groups, out of a total population of about 1400. I have not at hand the exact number of families represented by this number, but it was about 55. Thus the situation in mental diseases, mainly dementia praecox, manic depressive insanity, and involution psychoses, was not unlike that of feeble-mindedness.

3. What is the situation in epilepsy? The superintendent of the Massachusetts State Hospital for Epileptics at Monson states in reply to a letter that there are practically *no* families of epileptics in his institution, numbering 1500 people, and a thorough search revealed 4 families of 11 people!—4 as against 60 in one institution, 80 in another, of the feeble-minded, and as against 55 in the psychiatric groups.

In my own private and hospital practice, which is extensive, I rarely see brothers and sisters who are epileptic. It does occur, of course, but it is not by any manner of means so prominent as in feeble-mindedness; not nearly so prominent as in neurasthenia, to go into another field.

This one fact is worth more, in my opinion, than a thousand coincidences of migraine, blindness, deafness, etc., in the near and remote ancestors of the epileptic. Linking this fact with the known pathology of certain types of epilepsy—organic injury—the conviction is strong that epilepsy is mainly an affair of the individual and not of the stock; that it represents injury, perhaps to a germplasm, and certainly to the individual, whether that injury be pre- or postnatal.

Since the foregoing was written, there has been a great interest aroused in epilepsy and many writers, both in the United States and Europe, have concerned themselves with the mechanism of epilepsy and apparently are omitting from consideration as important, heredity. This is exactly what has happened with many another matter of science. As new facts are unearthed, old beliefs drop out of existence without much controversy or disproof. *They merely become obsolete.* The older point of view was that the epileptic attack was due to an excitation of the cortex of the brain, though from Hughlings Jackson down to our own times, there have been those who

disputed this. With the work on decerebrated animals, which was carried on by a long series of brilliant investigation, and especially, by the neurophysiologists of England, headed by Horsley and Sherrington, a new explanation for the epileptic attack has gradually evolved. This explanation is that the epileptic attack is primarily a decerebration, that is, a dropping out of the cerebrum, and especially the motor cortex, from activity, and thus releasing into excessive activity, lower motor centres, probably those of the midbrain. This is the point of view expressed by Kennedy (102), Rosett (163), and others.

Kennedy believes that there is a blanching of the cortex, that is, a deprivation of the cortex of blood at the beginning of an epileptic attack. Rosett especially has built up an elaborate theory of the epileptic attack, to which I refer the reader. Rosett believes that there is a constitutional basis for the epileptic attack. Experimental epileptic attacks have been produced by workers in the past and lately by Elsberg and Stookey (51), who found that convulsive attacks in animals could be produced by the injection of oil of absinthe, shutting off the cerebral circulation and other procedures. Especially interesting is their statement that the attacks, which are due to cerebral anemia, can be prevented by the circulation of Ringer's solution in the brain.

The important question—what is it that "decerebrates" the patient so that the lower centers get the upper hand, is entirely a clinical problem. We know that syphilis, tumor, cerebral arteriosclerosis, results of trauma, certain toxic states, produce epileptic attacks. There remains numerically the largest section of epilepsy in which we find no cause whatever. Studies in blood chemistry have proven unfruitful (Lennox, 119) for the most of the workers, but Osnato (150) claims that recent Italian work done by Cuneo, shows that epilepsy is due to a defective metabolism of starches. The term idiopathic epilepsy, as stated before, is given to those cases of epilepsy for which no adequate cause can be found. The term "idiopathic" entirely begs the question and should be dropped from the nomenclature of medicine, and the term "epilepsy of unknown origin," substituted for it. Meanwhile it seems proper to state that the hereditary factor in epilepsy has not been proven to exist; that there seems to be in many cases a constitutional factor, but this constitutional factor may rise de novo in the life-time of the individual himself from his uterine existence onward.

CHAPTER VI

Feeble-Mindedness

When the question of feeble-minedness is raised, the one thing that has become prominent in the mind of the community through the publications of the past twenty years and more, is that it is a menace to the world because it is strongly hereditary, and because the feeble-minded are prolific beyond the normal. Everywhere one hears of the feeble-minded criminal, the feeble-minded pauper, the unemployed feeble-minded, the feeble-minded prostitute, until it seems to some, undoubtedly, that crime, poverty, unemployment, prostitution and the like are problems of psychiatry rather than of sociology. In this last phase of feeble-mindedness, namely its relation to great social problems, I do not intend to go, but will content myself with the statement that this tide of publication and opinion is receding, and while some part of these problems is wrapped up with feeble-mindedness it is a small part and not the most important. Harm can only come to psychiatry and its functions in society if it makes claims ahead of the facts, if it penetrates into the problems of life more rapidly than it builds solid lines of communication.

I. THE NATURE OF FEEBLE-MINDEDNESS

The term feeble-mindedness is like the term insanity, used as if it were a unity of some kind, existing by itself as a pathological type of mind. This is as untrue of feeble-mindedness as it is of insanity, in that feeble-mindedness is a name given to a symptom or symptom complex existing in connection with a host of diverse symptoms and arising from very many causes. Therefore, to speak of the inheritance of feeble-mindedness or its relationship as a hereditary factor to other mental diseases it is necessary to discriminate, to say that one means the feeble-mindedness associated with organic brain disease of an acquired type, like meningitis or encephalitis, or birth injury, or the feeble-mindedness associated with disease of the thyroid gland, or the type of feeble-mindedness known as Mongolian idiocy, etc.

Feeble-mindedness, as a symptom, is a congenital or early acquired lack of mental ability, manifested by an incapacity or diminished capacity to remember, to learn, to carry out the functions of mind in the degree that we recognize as normal. Here enter many difficulties—some people think genius is abnormal and there is no definite agreement on what is normal in mentality. Though there are emotional and volitional defects in feeble-mindedness, though there may be character difficulties, the essential mental change is a deficient intelligence.[1] Until lately we had no means of definitely standardizing tests or results but in the last ten to fifteen years tests have been evolved, the age level tests, (Binet, Simon, Terman, Yerkes, etc.) which permit of more exact appraisal of the intelligence level. It is true that these tests are open to criticism, that they are not, as is claimed, tests of *capacity* but rather are tests of *knowledge;* it is true that their use has opened the flood gates of half baked, dillettante and cock-sure work on feeble-mindedness and intelligence,[2] but they are not to be condemned for these reasons. The tests are very useful, but they bring us into touch only with the *obvious defect* in feeble-mindedness and not with its real nature. The intelligence defect, which indeed is critical since without it we are not justified in the diagnoses, is in at least a large part of feeble-mindedness merely incidental, is a symptom like other symptoms, and related to causes to which I now turn.[3]

1. Trauma at childbirth, causing hemorrhage in the meninges, brain injury, and often associated with hemiplegia, diplegia, etc. Recently I examined 40 such cases at the hospital for feeble-minded in Waverly, Massachusetts. The only possible heredity involved

[1] No matter how lacking in emotional control an intelligent man might be no psychiatrist would call him feeble-minded, though as a matter of fact emotion is as much mental as intelligence.

[2] Hexter, M. B., and Myerson, A. This is a rather severe criticism of the work of Brigham concerning the intelligence of the immigrants. These authors contend that the intelligence tests as used in the army should not have been applied to the recent immigrant and that the conclusions drawn from them are not scientifically based.

[3] Therefore no intelligence tests, and no psychological tests, are the essential criteria for feeble-mindedness, though they may give us better means for measuring the mental damage, just as an engineer who told us how badly a locomotor's engine was wrecked could by this alone give us no real knowledge as to the cause of the wreck.

in such situations are the type of pelvis of the mother, or the overlarge head of the child. Quite often the causative factor is the unskilled application of forceps of the accoucheur. Since the functions of the brain are in part localized, though in an imperfectly known manner, a lesion in the certain areas will, if extensive enough, cause feeble-mindedness in any one.

2. Infectious diseases of childhood; encephalitis, meningitis, often associated with hydrocephalus cause feeble-mindedness, sometimes with epilepsy. Since serum has been used in epidemic cerebrospinal meningitis less of this type of feeble-mindedness has resulted.

3. Myxoedematous idiocy, cretinism.[4] In the United States there are few cases of familial cretinism, the disease occurring in isolated individuals without any definite heredity. As is well known the essential cause is a disease of the thyroid gland, resulting in hypofunction of the gland, which in its turn leads to (*a*) stunted growth, to an extreme degree, (*b*) certain changes in face, hair, tongue, hands, etc., of characteristic nature, resulting in the caricature in man, the cretin, (*c*) idiocy, usually of a low grade.

What is here emphasized is that the idiocy is *incidental*, and biologically is not the important change, which is the thyroid gland deficiency. Further this disease has no essential relationship to the feeble-mindedness of trauma, or the hereditary type. Though classed as feeble-mindedness it is essentially a problem of endocrinology.

4. Mongolian idiocy. This peculiar type of defect is associated with a remarkably constant Mongolian type of facies, dwarfism of extreme degree, and a low grade of mentality. The Mongolian idiot usually comes of a normal familty, is rather often the last member of such a family, but aside from that there is no general evidence of any abnormal heredity. Something happens, perhaps to the endocrinal glands of the individual. What has been emphasized in the case of cretinism is true here, namely, that the mental state is only part of a generalized damage affecting the organism as a whole. It will later be seen, that this is true of the familial types of feeble-mindedness as well.

[4] Even F. Lenz, enthusiastic Mendelian, has absolved this disease, and Mongolian idiocy, from hereditary implication. My own studies in nearly 50 cases show no hereditary relationship.

5. There is also a group in which gross failure of brain development, or gross failure of the organs of sense, has brought about idiocy. These individuals remind one of the monstrosities[5] that biologists obtain by subjecting eggs to various chemicals, and indeed they belong in the field of teratology rather than in psychiatry. They are simply viable monsters, there is usually nothing whatever in their antecedents to account for them. True, one of their parents may have been alcoholic, and an uncle may have epilepsy—but these are not explanatory phenomena, they occur too frequently to account for such rarities. The fact that similar caricatures on life can be created by experimental toxic influences working in healthy eggs suggests strongly that something of this nature is responsible.

6. Syphilitic feeble-mindedness. Just how large a proportion of feeble-mindedness is due to syphilis is a matter concerning which there is much contradiction in the estimates.[6] Here as everywhere it is difficult to separate coincidence from cause. Because the parents have syphilis does not prove that the mental defect of the child is due to that disease. Though there are a few well recognized signs of congenital syphilis various authors have described minute phenomena as also diagnostic of syphilis. The trouble with these signs of degeneracy is that they are far more common than syphilis itself and it is difficult to establish any causal relationship. Further, the Wassermann reaction is very uncertain after the fourth or fifth year of life in the congenital syphilitics, except where we deal with

[5] See Stockard's article, American Journal of Anatomy, 1919.

Stockard makes the point that all types of monsters are developmental arrests and due to environmental changes. He gives a long list of methods and techniques by which this can be brought about and compares it to feeble-mindedness in a very pertinent manner.

Also, Féré, Charles, La Famille Neuropathique.

[6] For a discussion of this important question see Solomon, H. G., and M. H. "Syphilis of the Innocent." Dr. Solomon is very cautious in his conclusions concerning the rôle of syphilis in feeble-mindedness. Because the general hospitals have children showing an average of 5 per cent positive Wassermann reaction, and in the schools for the feeble-minded they show only 6 per cent, he concludes that "it is not possible to state, as is so often done, that congenital syphilis is a great element in the production of feeble-mindedness" but says "on the other hand, there seems to be little chance for doubt that certain individual cases of feeble-mindedness owe their origin to syphilis in the parent." Other observers whom the Solomons quote have, on the other hand, through different kind of study, that is through the study of the mentality of known syphilitic children, come to the conclusion that syphilis causes a good deal of backward mentality.

congenital tabes or paresis. It is therefore difficult to state, except in definitely-diagnostic cases, that syphilis has caused feeble-mindedness. These definite cases constitute from 1 to 4 per cent of hospital cases and are frequently familial, i.e., occur in two or more siblings. Possibly this is a minimum estimate, and there occurs possibly a number of lightly affected individuals, blighted personalities, whose condition cannot definitely be ascribed to syphilis.

7. We come to a large mass of the feeble-minded, who do not present the ordinary signs of organic disease of the brain, in whom family mental disease is prominent and who present the bulk of the problem of feeble-mindedness. What is the background for these types, and how are we to evaluate the heredity that is almost universally postulated concerning them?

In this book on feeble-mindedness Goddard (66) decides that feeble-mindedness *or the liability to become feeble-minded* is a Mendelian trait. He cites some 100 cases, in which family studies have been made in the way discussed before this. Goddard and Davenport are in full accord as to the traits they regard as neuropathic and as to the value of the technique they employ in obtaining their data. The keystone of the arch of their results and laws is the field investigator and her surmises as to the mental and physical state of the dead and the quick; and the cement is the theory that thirty or forty different conditions are neuropathic traits and due to the lack of a unit character.

I cite as an example of Goddard's (65) investigations the famous Kallikak family, or rather the famous account of the family, quoted in all the lay literature and held up as the spectre of the threatened predominance of the feeble-minded. No royal family has enjoyed quite such a prestige as this group, except their associates in notoriety, the Jukes and the tribe of Ishmael.

The book has all the dramatic flavor of the missionary spirit, or of one who "views with alarm" and wishes to awaken into vigilance the threatened normals. It opens up with a long, dramatically necessary, scientifically superfluous account of a female Kallikak in Goddard's[7]

[7] Curiously enough it was a very unsophisticated-looking girl student who suggested to me that it might well be that Martin Kallikak was not the father of his alleged sub rosa feeble-minded descendants, that no one knew who else might have mated with the "nameless feebleminded girl."

As a bit of worldly wisdom this deserves the attention of Dr. Goddard and his field workers.

institution at Vineland, and after regaling the reader with her deficiencies and ordinary feeble-minded qualities, the author depicts with gusto, and graphically, the horror he, and his field-worker, felt as they traced backward from Deborah, through a long line of defectives, to the union of Martin Kallikak and a "nameless feeble-minded girl" in the days of the American Revolution. Quite considerately Martin Kallkikak performed an experiment for the sake of the writer of the book and the rest of society; he united himself first with this "nameless feeble-minded girl" and started this long row of degenerates—feeble-minded, syphilitics and alcoholics —and then, reforming, used his germplasm in orthodox fashion by marrying a nice girl who bore him nice children and started a row of nice people—all nice, no immoral, no syphilitics, no alcoholics, no insane, no criminals—as perfect in its way as its sub rosa begotten half-relatives were imperfect.

I confess to a feeling of shame in the presence of the work done by the field worker in this case. I have had charge of a clinic where alleged feeble-minded persons were brought every day and I see in my practice and hospital work murderers, thieves, sex offenders, failures, etc. Many of these are brought to me by social workers, keen intelligent women, who are in grave doubt as to the mental condition of their charges *after months of daily relationship*, after intimate knowledge, and prolonged effort to understand. Many a time it has happened that one of these excellent women has declared that her charge must be feeble-minded or insane, and yet the mental tests and psychological examinations have shown the contrary, that the patient was of full average mentality or better; often it has happened that the social worker (or the informant) has believed that the "social problem" was not feeble-minded, and yet the thorough examination has disclosed undoubted feeble-mindedness. And I have to say of myself, with due humility, that I have had to reverse my first impressions many and many a time.

Judge how superior the field workers trained by Dr. Goddard were! Not only do their "first glance," tell them that a person is feeble-minded, but they even know, without a shadow of doubt in so far as the book intimates, without the faintest misgiving, that "a nameless girl" living over a hundred years before in a primitive community, is feeble-minded. They *know* this, and Dr. Goddard acting on this

superior female intuition, founds an important theory of feeble-mindedness, and draws sweeping generalizations, with a fine moral undertone, from their work. Now I am frank to say that the matter is an unexplained miracle to me. How can one known anything definite about a nameless girl, living five generations before, of whom there can be no records, whom no one remembers, whom no one has seen? Granting for the sake of argument that perhaps she had a mole on her left arm above the elbow by which she may have been identified by her contemporaries, how can any one know that she was feeble-minded? I cannot get any definite information about my great-great-grandfather, much as I have tried—but a girl who left so little impression on her times as to be "nameless" is positively declared to be feeble-minded. It seems to me that the elaborate and excellent psychological tests used (and in part evolved) by Dr. Goddard might well be declared superfluous in the face of such transcendent knowledge.

(There is a certain insolence in declaring that the same kind of knowledge "by which we know of Washington and Lincoln" is used to determine the character and mentality of the Kallikak family. True a rush light and a huge search-light are both forms of illumination, but a rush light would give no information about a distant object. On the mighty figures of Washington and Lincoln there played the search-light of their position in the eyes of their comtemporaries, the search-lights of hate and love, of the headship of a great nation. Eager historians have given their lives to their study, documents upon documents have been found, disputed and evaluated, and their name appears emblazoned on history's page in the glare of many search-lights—A nameless girl, companion of an amour of a Revolutionary soldier, some unknown tap-house habituée, is declared feeble-minded on the kind of knowledge we have of her contemporary, Washington!)

I wonder what a syphillographer, a medical man who has studied years in the medical school, gone into hospital work, taught in a university, and who with all his experience makes a diagnosis of syphilis on a patient only after careful examination and the Wassermann test, would think of the diagnosis of syphilis so glibly made of people dead—gone many years, or of people talked about for fifteen minutes, or perhaps even talked to? Throughout the history of the

Kallikaks this diagnosis is made, without the possibility of evidence that would be admitted anywhere as having any value! And even if it had any value, even if there were syphilis in the Kallikaks, just what would that signify? Syphilis is an infection, found in all ranks of society, amongst gallant soldiers, shrewd men of commerce, in the ranks of the great writers, and even amongst those who dare to preach to their fellows. Syphilis is an infection, can be transmitted to experimental animals whose ancestors were not psychopathic, and represents more an accident than anything else. I repeat, there can be no ground for the diagnosis as made by the field worker but even if she conducted a telepathic Wassermann laboratory and could diagnose the disease on the dead and absent it would have but little significance.

It is obvious that if one is wedded to a Mendelain theory one must have numbers to bolster up that belief, and there is a temptation to make out as feeble-minded and the equivalent whatever even roughly looks like it. It is true that Goddard says that the Kallikaks are high-grade morons, not imbeciles or idiots, *but it is just exactly these concerning whom a diagnosis cannot be made on history or hearsay;* it is in the high-grade moron on whom a real and thorough personal study must be made, the kind of thorough study quite typically done in Vineland.

Further, if the Kallikaks were entirely as represented, they would be too exceptional to be of value. I doubt that the field worker looked with zeal and energy for cases of feeble-mindedness and syphilis, mental disease, etc., in the legitimatized descendants of the sex offender Martin Kallikak. (Strange that this trait did not crop out in these normal descendants. Does marriage protect so well as that?) Every neuro-psychiatrist sees many cases of mental diseases arising de novo from "good" stocks, and every day experience tells of listless, good-for-nothing descendants of energetic, worthwhile ancestors, just as the muck of the earth (as it is nowadays the American (?) fashion to speak of the immigrant) breeds genius and character. Breeding the human-being is still an unknown science, and nowhere in real life does it run so true to form as depicted in the history of the Kallikaks.

In ethics two wrongs do not make a right, and in science a thousand instances of guess work, intuition, snap judgments and hearsay will do good neither to the Mendelian theory nor to eugenics.

Fernald's studies (54). In relation to the fecundity of the feeble-minded the reader is referred to Dr. W. E. Fernald's report on the discharged Waverly patients. There were discharged from his institution in twenty-five years (1890 to 1914) 1537 patients, of whom 646, 470 males and 176 females, were in the community and could be studied. These 1537 people were, in the majority of cases, dismissed under protest, as it is the policy of the school to insist on permanent segregation.

Of the 646 people, 82 had died, and 101 were readmitted to the school. Of the 176 females 27 had married and there were as issue 50 children, and of these 17 had died leaving 33 children, which hardly looks like the "remarkable fertility of the feeble-minded." Seven of the married women had no children. Nearly all of the married women had married men above their own social status and that of their own parents, i.e., these feeble-minded women had advanced socially.

Of the 27 married women 11 had done well, and 16 had behaved badly, being sexually promiscuous, etc.

Of the 176 females there were 11 unmarried mothers, with 13 illegitimate children. None of these had been supervised and most were returned to the hospital. "Apparently the discharged female patients have not contributed largely to the sex and venereal problem, but this is undoubtedly due to the school training and the supervision of relatives. In fact, of the 90 discharged females now in the community 52 are apparently giving no trouble, though needing supervision.

Of the 470 males, 13 had married and have 12 children. No comment necessary. "There was a surprising small amount of criminality, sex offence, and especially of illegitimacy."

I mention without especial emphasis that the children of these feeble-minded people seemed normal. As I understand from Dr. Fernald this statement is made on school record, history and an interview by the social worker. It would have been better to have examined these children medically and psychologically. Yet the evidence for their normality is far better than the evidence usually submitted as conclusive of feeble-mindedness.

Since feeble-mindedness is a congenital condition and since the majority of these known descendants of the feeble-minded are not feeble-minded but appear and act in a normal manner it is obvious that not all feeble-mindedness perpetuates itself. Further, the

great prolificness of the feeble-minded is hardly in evidence here, since the families of these feeble-minded are small, even if we include the illegitimately born children. What is really meant by the talk of the *fertility of the feeble-minded* is the *fertility of people of low cultural level*, of low economic status, or else unsophisticated in the trends of modern society.

In the people I know the best, the Jews, this is remarkably shown. In the Ghettos of Russia, Poland and Roumania, the Jew married at from eighteen to twenty-one, and had 4 to 6 children, or more. And the Jews who immigrate to this country, or any western European country, continue to have large families, and to marry young. But the second generation, American born,[8] marries late, and has so few children that if immigration is cut off the Jewish question in America will settle itself by race extinction. It is the advance in luxury, in urbanization, in economic and social status, that lowers birth rate, not increase in intellectual level. It is late marriage and "knowing how" that brings about the one or two child family, and the western world is rapidly getting to the point where late marriages and "knowing how" will be quite the rule whether or not legislatures practicing what they do not preach say yea or nay.

At this point I shall make a preliminary statement concerning the feeble-minded families now at Waverly. By this I mean the families of siblings at present within the hospital (in one case including a mother as well as her children). Since the families have not been thoroughly studied with regard to their ancestors and relatives—thoroughly from my point of view—and since the number (60 families) is too small for statistical elaboration, I shall content myself with presenting a few facts.

1. In a large part of the families the siblings present so low a mental level (idiots and low-grade imbeciles) conjoined with so much physical inferiority that the males could not have sexual intercourse, and it is doubtful if the females are fertile. Even if fertile it is doubtful whether they would be subjected either to seduction or rape. In other words, in these familial cases there is a strong tendency to race extinction due to the very low *physical and* mental level of the members of the families. This is also true of the non-familial cases—it is rare to find a very low mental level (idiot or low imbecile) conjoined with any real vitality or fertility.

[8] For an interesting discussion of this point see Morris Fishberg (56).

2. Many of these families of siblings show their inferiority in a gross physical disorder as mental disorder, and no medical man can view them without the distinct impression that the "feeble-mindedness" is a symptom in the course of something which has blighted or injuried the whole organism. Defective heads, defective faces, defective teeth, and especially defective jaws, defective stature, anomalies of make-up, queer hands, extraordinarily poor feet, together with, in a few cases, absence of eye-balls, inability to sit, stand or walk are as conspicious as the defective mentality, and there is no more reason, logically, to select the term feeble-minded to designate the worst of these cases than there is feeble-jawed, feeble-feet, etc. It is notorious that the lower the mental level the greater the degree of physical defect, though there is by no means an exact parallelism.

3. In many cases all the siblings of a generation were in the institution or definitely feeble-minded. In others some of the siblings were apparently normal. It would take a study, such as is now in project, of all the feeble-minded families of the state to make any statistics worth while.

4. In many cases there was very obvious mental defect in the parents. In other cases the parents were in the main normal. I do not consider "alcoholism in a brilliant man" as explanatory of the idiocy of his children, nor do I regard as important the fact that the mother had migraine. I refuse to be impressed unfavorably by the family history because a grandfather was deaf or because another was for a short time in his youth an offender against the law's majesty. I scoff at the implication that because the "paternal great grandfather and great grandmother were first cousins" that we have any clue whatever to the gross defect of their great grandchildren, their father and mother being "superior people." I am not at all convinced that the chorea of the mother's childhood can in the present state of our knowledge account for the injured physical and mental state of 2 of her 6 children.

As I said above feeble-mindedness apparently occurred in much too large a proportion of the ancestors of these siblings to be accidental. We have then the undoubted fact that some forms of feeble-mindedness tend to run in families, but we still have as unknown the explanation of its origin since we see it arise de novo in normal groups, arise and become familial, for I exclude now the non-familial types of feeble-minded, cited in the earlier part of this

chapter. Further, like mental disease of other types, as we shall see later, it tends to become extinct, for obviously there is one trend towards such inferiority as to make a new generation impossible, just as there is undoubtedly a trend towards recovery of the stock, as shown by Dr. Fernald's cases.

Since in these families which I am now so sketchily presenting there are three syphilitic groups, the question of germplasm injury (as against hereditary character in the sense of spontaneous variation, Mendelian unit) arises. Frankly, I am prepossessed in favor of germplasm injury, and will discuss the whole subject in Part III of this book. As I see these patients I am convinced that they are injured by some agent, and this injury is communicated for one or more generations, growing worse in some cases, better in others. This neo-Lamarckianism has some remarkable experiments to substantiate it, as I shall show later.

I have not attempted to do justice to the great literature on feeble-mindedness. The opinion of a few other authors may be of interest. Martin W. Barr (7) in his text book "Mental Defectives" lays great stress on heredity of feeble-mindedness, but cites statistics like this. "Defective mental capacity in one or both parents 65 of 185. Consanguinity in parents 20 cases. *Tendency to tuberculosis, scrofula or eruptive diseases 41 of 145.* Fright or grief to mother at time of conception, etc." When statistics are cited in this manner one is frankly justified in discarding them entirely. Surely a tendency to eruptive diseases has no place as a hereditary cause of feeble-mindedness. A. F. Tredgold (199) cites the work of the Royal Commission which was appointed to investigate feeble-mindedness, and amongst whose members were such distinguished men as Allbutt, Asby, Mott, Crichton-Browne, Reid, etc. This commission found that feeble-mindedness was "spontaneous in origin and tends strongly to be inherited." Tredgold follows Morel and Clouston in believing in degeneracy, and makes very interesting observations that feeble-mindedness results from a diminished germinal vitality, in consequence of which development tends to be incomplete. "This lessened potentiality is especially marked in that constituent which determines the development of the central nervous system—the neuronic determinant—but it is often more widespread and then affects other tissues of the body also. In other words, the inheritance takes the form of a neuropathic diathesis; the actual manifestation of this innate weakness, however—that is the form it assumes—being often

dependent on the nature of the environment. In short, I regard the germinal variation present in these persons as a pathological one, as being in the nature of a vitiation." Thus this author does not regard feeble-mindedness as a hereditary character as much as he regards it as a germinal disease character. Shuttleworth and Potts (176) in the book "Mentally Deficient Children" cite 14 results of the Wassermann reaction in feeble-minded children, the percentage of positive Wassermanns ranging from 1.5 per cent of Thompson, Boas, and their co-workers, to 60 per cent of Fraser and Watson. Most of these papers they cite were published in 1911, at which time most of the Wassermann work done was not worth the paper it was published on because the technique had not been standardized. Since that time it has been standardized and Wassermann work is worth while.

Mott, quoted by these authors, believes that syphilis is an active agent in the production of congential weakness and degeneracy. "It must have," says Mott, "a devitalizing effect on the offspring that survive." In this he agrees with William W. Graves of St. Louis, who says that syphilis makes a good heredity bad and a bad heredity worse. L. P. Clark and Charles E. Atwood writing in 1922 are very skeptical of the data on heredity of feeble-mindedness. They cite Wilmarth and Southard as showing the comparatively large number of cerebral disease in autopsies on the feeble-minded. Thus in 70 such autopsies Wilmarth reports nearly 75 per cent as showing gross cerebral lesions. Cohen and Clark state the fundi in feeble-minded show much actual change, from which they conclude that the neural side of feeble-mindedness is fully as important as the psychic side. Clark and Atwood are inclined to believe that alcohol and syphilis injure germplasm and that such injury is responsible for a great deal of mental defectiveness. It is implied throughout their writing, though not explicitly stated, that germplasm injury or actual cerebral organic injury to the individual is responsible for most of feeble-mindedness. I forebear citing more of the literature on feeble-mindedness. Certain of the larger aspects of the situation will be discussed in a later chapter. The trend of opinion is certainly away from the conception of feeble-mindedness as a unitary character. Most writers are firmly convinced that there is a familial transference of certain types of feeble-mindedness, and there is very distinctly appearing the belief that these familial cases originate in injury to the germplasm.

CHAPTER VII

Five Important Mental Diseases

There is a group of mental diseases with which this book mainly deals inasmuch as the bulk of its original work concerns these diseases. I turn the attention of the reader to five mental diseases, though it is just to say that the term "disease" is prematurely applied, since we have not yet the understanding of them which is usually implied by "disease." Our pathology is scanty, and on the whole not fortified by control or solidified by confirmation; our diagnostic criteria are unsatisfactory, and even the names of the diseases are at present under fire.

These five conditions, which is perhaps a better term than disease, are dementia praecox, manic-depressive insanity, paranoid psychoses, involution diseases, and senile dementia. To describe these conditions in detail would mean the writing of a text book on psychiatry, and so I shall outline them laying most stress on the chief clinical appearances. Because the cases cited in the second part of this book are taken from old and new records of the Taunton State Hospital, I am obliged to make the rough classification above given, which is good enough in the present state of our subject.

Dementia praecox. This, probably the most common of the mental diseases,[1] starts mainly in early life and in the majority of cases proceeds leisurely or rapidly to what has been defined under the term dementia. While this terse account corresponds to perhaps the greater number of cases, it is also true that there are many cases in which the disease *apparently* starts in middle life, in my opinion even later; there are many cases which never reach the asylum and which do not incapacitate the patient from carrying on as citizen and worker. Though it is felt by most psychiatrists that dementia praecox patients do not recover except in rare instances, this

[1] Discussing the age distribution of 1054 cases of dementia praecox, Kraepelin states that 3.5 per cent occur at ten years, 2.7 per cent at fifteen years, 21.7 per cent at twenty years, 22.8 per cent at thirty years, 13 per cent at thirty-five years, 5 per cent at forty years, 3.3 per cent at forty-five years, 1.2 per cent at fifty years, 1.1 per cent at fifty-five years, and 0.2 per cent at sixty years.

is probably more true of the asylum cases than of the community cases. In private practice one sees patients who seem to be drifting towards the asylum, but who, without known cause, make an adjustment and, at least, *seem* to drift back into the harbor of sanity.

That this variability in age of onset, course, and symptoms of dementia praecox is not exceptional in disease needs only a few examples to prove. Tuberculosis is extraordinarily variable in all these characters, and so is syphilis. In tabes dorsalis, e.g., the range of symptoms is from cases with only pains and aches to those with the most disintegrating phenomena of bones; the course ranges from acute onset and acute ending to a stationary type of disorder lasting a half century.

The four main forms of dementia praecox are the paranoid, the catatonic, hebephrenic, and simplex.[2] The paranoid type is characterized by the slow evolution of ideas of persecution, usually fantastic, and is usually associated with hallucinations of one kind or another. In the course of time dementia occurs, the delusions vanish, apathy and general mental disintegration become the outstanding facts.

In the catatonic type, which merges with the other groups, and often becomes identical with them, there is usually a sudden onset, perhaps marked by violence and great purposeless activity, and later by negativism, cerea flexibitas, repetition of movements and phrases, etc. Ordinarily there is a basis of delusional thought with intense emotional disturbance, which perhaps becomes more manifest after the acute onset is over. Sometimes the catatonic type makes a "recovery" which may last for a long time, but usually such a recovery is incomplete, and gradually gives way to an emotional, intellectual and volitional deterioration.

In the hebephrenic type there is a long prodromal stage of character peculiarity, then a sudden efflorescence of delusions and hallucinations, with conduct disorder and lowered intellectual capacity, then comes apathy and rapid mental deterioration. This is usually a condition of yearly youth and is very common in the syndrome of dementia praecox.

[2] In the latest Kraepelinian classification there are 11 forms of the disease. That dementia praecox is an entity is vigorously denied by a large and influential part of American psychiatry, and the French school has not on the whole enthusiastically accepted it. Nevertheless it still represents a better concept than anything its assailants have advanced.

The simplex type is characterized by a gradual falling off of mental ability and a steady loss of interest in the rest of the world, with but a meager content of delusions and hallucinations. It is more like feeble-mindedness than any of the other forms of dementia praecox, but differs from it mainly in the fact that the deterioration takes place after a more or less normal development has been reached.[3] The term "more of less normal" is used advisedly, since in many cases the patient has always been recognized as queer, odd, unsociable, of low mentality, etc. In a later chapter facts bearing on this will be presented.

This little outline cannot do justice to the rich efflorescent symptomatology of dementia praecox. One goes into a ward—one sees a patient stalking haughtily to and fro, muttering to himself, eyeing his neighbors with suspicion and disdain, or smiling with unconcern for those around him. Questioned, one finds that he believes that every one talks against him, that his rooms are flooded with gases, his food is poisoned by monkeys in the employ of vaguely identified people, that he hears and sees queer things of all kinds, that he believes himself in a vague way to be some great personage, and is beginning to be confused and deteriorated. His language, vague and difficult to follow, is filled with symbolisms and neologisms intelligible only to himself—a paranoid dementia praecox.

Or one sees in a corner a patient, standing with head down, and limbs in a grotesque attitude. He does not answer when addressed, but resists violently if any effort is made to change his position, or perhaps retains any position, no matter how grotesque, into which he is placed, a catatonic dementia praecox.

On a bench in a ward is a curled up figure, lying motionless, like a bag of meal, taking no interest whatever in what goes on, drooling saliva, wetting and soiling himself, eating if he is fed, and sleeping if he is put to bed—in a word, vegetating—a hebephrenic.

In the fields of the hospital, in the barn, doing little odd jobs here and there are dull looking persons, with silly smiles and vague monosyllabic answers, doing what they are told in an inferior way, masturbating, obscene, often filthy. These are the simplex praecox cases.

[3] As before stated Kraepelin (108, 109) believes that some cases of feeble-mindedness are really congenital dementia praecox.

But these are hospital types. In the community are all kinds of personalities, paranoid, stubborn, hypochondriacal, unsocial—whom the psychiatrist recognizes as essentially undeveloped cases of praecox. Moreover in the early stages of the lives of those patients who eventually come to the asylum there are neurasthenic and psychasthenic phases that strongly resemble true neurasthenia and psychasthenia, and one who later is to become apathetic and demented is horribly anxious, tormented by doubts and fears, given to endless cogitations about himself and the world he lives in. One sees many cases, as I have before stated, which seem to be headed fully towards dementia praecox, which emerge into quasi-normality and remain there. One is impelled to believe that there is in addition to the pre-dementia praecox stage described by Hoch, Meyer and Jelliffe (82) as abortive praecox in which recovery takes place before definite breakdown takes place. In other words the hospital cases are really end stages of the non-recoverable cases, but in the community are many other types which never reach the hospital because they never need to.[4]

Whatever the source or origin of this disease it is not known. True there are two great groups of explanations, which are, however, poorly grounded and without definite proof.

There is the psychogenetic school, which finds the origin of the disease in conflicts, personality trends, etc. The school of Freud, though it has really done but little work on this disease, having paid its main attention to the psychoneuroses, has no hesitation in finding homosexual, narcissitic, oedipus-complexes of all kinds, and reversions to foetal states. Bleuler who is willing to accept a bodily disturbance behind the psychic disturbance makes a characteristic explanation. The individual falls, through conflict between

[4] A valuable booklet is "Dementia Praecox," containing three articles by August Hoch, Adolf Meyer, and Smith Ely Jelliffe. These authors criticize the Kraepelinian concept of dementia praecox and substitute for it as a disease the conception that "it is possible to formulate the main facts of most cases in terms of a natural chain of cause and effect, utilizing the psychobiological material at hand, better than a dogmatic assumption of a specific but hypothetical unitary toxic principle" (Meyer). The psycho-dynamic school thus postulates personality types of a peculiar kind (shut-in type, e.g.) which gradually develop in the symptoms of mental disease as a result of interreaction with the environment, the environment meaning of course the social restrictions and injunctions more than the purely physical facts of life. Starting with difficulties in adjustment the individual passes in a dynamic manner to mental disintegration.

desire and reality, into what Bleuler called[5] autistic thinking, creating a world of phantasy in which he prefers to live, but into which the real world persists in obtruding itself, so that delusions and hallucinations appear. Bleuler's[6] term schizophrenia, indicating the splitting up process that takes place in the personality, is now classic and in many clinics has superseded the term dementia praecox. Elsewhere I have voiced the opinion that the splitting up process is no more a disease than "inflammation" is, but like it names a pathological process operating in many diseases, probably in all mental diseases. Jung has psychoanalyzed cases of dementia praecox and finds, behind the irrelevant and deluded-thinking, typical Freudian complexes. He is, however, willing to concede that an auto-intoxication may condition the psychological picture. Hoch (81) and Meyer (132, 133, 134), in this country, as well as many German workers, have elaborated the idea that there are types of personality predisposed to the disease. Especially do they emphasize the shutin personality as peculiarly predisposed, a non-social type out of contact with its fellows either through feelings of inferiority or superiority, seclusive and building up an unreal inner world which finally becomes pathological.

In regard to the theories of Freud, Kraepelin says this:

> I must frankly confess that with the best will I am not able to follow the lines of thought of this "metapsychiatry" which like a complex soaks up the sober method of clinical observation. As I am accustomed to walk on the sure foundation of direct experience my Philistine conscience of natural science stumbles at every step on objections, considerations, and doubts, over which the lightly soaring power of imagination of Freud's disciples carries them without difficulty.

The illustrious discoverer of dementia praecox further states that it is possible and probably true that the character peculiarities so commonly observed in the early life history of the person who later develops dementia praecox are probably symptoms of the disease rather than the basis upon which it develops. This seems to me the correct view of the character peculiarities. Those who wish to understand dementia praecox in detail may turn with advantage to the book of that name written by Professor Kraepelin.[7]

[5] *Was called,* it is now labelled vereistis.

[6] Bleuler's book, Schizophrenia, is a classic and very valuable to psychiatry.

[7] Dementia Praecox, Emil Kraepelin, translated by Barclay and Robertson, 1919.

PATHOLOGY

As to the pathological changes the researches of Alzheimer (183) Klippel (183), L'Hermitte (183), De Buck (183), Deraubaix (183), Zingerle (183), Zalplachta (183), and Southard (183) make it plain that lesions are found in dementia praecox which chiefly affect the deep cortical layers and are associated with gliosis.[8] The question as to the relation of these changes to the disease is unsolved. Their existence can be admitted without necessarily ascertaining a causative relation. It cannot yet be told whether they are incidental to catatonia, excitement, emaciation, tuberculosis, etc., or whether they are etiologic. As regards the clinico-pathological changes, it seems to be established by the work of Hielman, Kruger (183), Purdum and Wells (183), Schultz (183), Itten (183), and others that there is a lymphocytosis and eosinophilia in dementia praeco particularly in the excited and catatonic states, and that there is often an increase of red cells. This blood picture is variable and disappears in the remissions and also in the old vegetative cases. There is a remarkable similarity between the white cell picture here described and that found in thyrogenous disease and there is some evidence of a disturbance in the internal glands. Thyroid therapy has in consequence been employed but without result. Kraepelin is still inclined to favor the theory of auto-intoxication with disorder in the sex glands, but admits that "convincing proof is unfortunately not available."

A development of possible importance in the last few years is the use of the Abderhalden reactions in the establishment of pathological processes in the glands of various organs, with the anouncement of Fauser that ferments (abwehr fermente) were present in the serum of dementia praecox patients which digested sex glands and cortex, especially in acute cases. Investigations have been made by Wegener, Fischer, Theobald, Neue, and others which agree in the main with Fauser, while Kafka, Mayer, and Golla cannot substantiate his findings. Orton (149) comes to the conclusion that

> Even if we accept the theory and the results of the most hopeful investigators we are only brought to the beginning of a wider field of investigation

[8] Every once in a while some one "discovers" that there are fat granules or acidophilic granules in the nerve cells in cases of dementia praecox. The only trouble with these discoveries is that a study of normal brain discloses an appalling amount of pathology of all kinds in them, as well.

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as by the interpretation of the theory the results speak only for a faulty metabolism in specific organs and as yet give no light on the underlying causes.

That the sex life of the patient with dementia praecox is very frequently abnormal is an old story. Many dementia praecox patients are hypoerotic and many more are given to masturbation, and especially the males. Sex delusions are exceedingly common and it is not at all surprising that the ancient idea of masturbation as a cause of mental disease arose. Indeed many a marriage is made with the totally erroneous idea as its basis that a normal sex life will cure a case of incipient dementia praecox. There are patients with dementia praecox who seem to have led a normal hetero-sexual life but these are rather exceptional. In the stage before the breakdown the patient lives in a sort of mental sex-stew, a phantastic wish-world where desire is satisfied without exertion or conquest.

This makes the recent work of Mott (139) the more pertinent and important. I cite here and there from his recent articles dealing with the condition of testes and ovary in dementia praecox.

After dealing with the psychologic and physiologic inter-relation of nervous system and sex organs, of emotions and purposes with sex feeling, he states that even in advanced age the seminal vesicles and spermatozoa show little or no defect, whereas even in early cases of dementia praecox marked degenerative changes are seen. "Dementia praecox shows much more degeneration than general paresis. The testes (in dementia praecox) show very little spermatogenesis." Examination of the ovaries show similar results to those observed in males. There seems to be a lack of specific vital energy of the reproductive organs in dementia praecox (and congenital imbecility). The ovaries are smaller and the surface is white and crinkled, the tissue is dense and there are a few corpora lutea.

Mott believes that in dementia praecox there "is an inborn lack of energy throughout the whole body but most manifest in the organs of reproduction." The regressive atrophy of the reproductive organs cannot be counted as a direct cause of the mental and bodily signs and symptoms of the disease. It must, however, be part of a degenerative pathological process.

This, it is evident, is quite different from the view which regards dementia praecox as a biological variant from the normal, as a Mendelian recessive or dominant character.

Heredity in dementia praecox will be discussed in Part II of this book, but whatever theory is used to explain dementia praecox must take into account the fact that in many cases there seems to be a familial transmission. This makes the psychological or temperamental genesis incomplete, for if the temperament is transmitted in the germplasm, then there surely is, from the medical or biological standpoint an organic basis, and the psychological or temperamental origin is entirely secondary. This likewise is true of the cortical changes, for these are important as phenomena, but not as causative, since we cannot assume that they arise by themselves. The changes described by Mott are strikingly similar to the changes described by Forel and Bertholet in chronic alcoholism; i.e., though no one considers alcohol as a cause of dementia praecox, some other blastophoric substance[9] may be at work.

I shall discuss the Mendelian theory in relation to dementia praecox later. Davenport and Rosanoff in America, Rudin in Germany, have been its principal proponents. But Mendelian inheritance is not an explanation as to cause, it is an analysis of occurrence and an effort to make the statistics of occurrence correspond with those of a Mendelian ratio. Therefore it is not especially pertinent to the discussion at this juncture.

Paranoid diseases. I am using this term to describe a group of diseases in which, essentially systematized delusions of persecution and grandeur arise in an individual who shows few other mental-disease signs of the hospital type, i.e., there is no deterioration, and there is complete or almost complete absence of hallucinations. I realize that this is a crude classification but as a matter of fact these are the criteria used to separate the paranoid diseases from paranoid dementia praecox. Further in analyzing hospital histories it is impossible to study the cases on any finer basis. The various groups of the paraphrenias, as described by Kraepelin in his latest editions, are over-refined separations from the main groups, they represent a sophisticated psychiatry which has no real legs to stand on.

There is no known pathology in the paranoid diseases, and there is no definite psychological explanation which is more than an account of its evolution. It is true that the Freudians have found homosexual roots to the disease, and there is now-a-days much talk

[9] As I have before stated Pilez intimates that syphilis is the blastophoric agent at work in dementia praecox.

of inferiority complexes which are compensated for by persecutory ideas and ideas of superiority. I must refer the reader to the compendious and stupendous psychoanalytic literature for this discussion. In any case it cannot bear on the hereditary transmission of the disease. The classical psychological explanation (Mendel, Krafft-Ebbing, etc.) viz., that we deal, in the case of the paranoid personalities, with variations in the self regarding and the social instincts, and that it is in the interaction of these defectively constituted individuals with a world which refuses to accept them at their own valuation, from which the particular symptoms arise meets my needs, at least, to a certain degree. If we consider temperament as the trend of instincts, emotions and intellect, and remember the wide variability of the characters of man, we can easily conceive that since temperament is organically conditioned, a variability of a pathological nature can be passed from one generation to another.[10]

The manic-depressive psychoses (*Cyclothymia*). This very common disorder, like dementia praecox, a synthetic product of German psychiatry, is characterized clinically by two main features:

1. It is periodic, occurs and recurs, has periods of normality interpersed between "attacks," and on the whole tends only slightly, if at all, towards a dementia. Though in the attack there may appear to be almost complete lack of mental functioning, in the recovery the return is almost to the normal, if not to the normal.

2. There is a change in mood, taking one of two directions; either a lowered mood, a depression, or melancholy called the depressive phase, or an excited, joyful, exalted mood, called the manic phase. In the classical cases the depressed and excited moods alternate either directly or with normal phases between, but the classical cases are very rare in my experience,[11] and in the experience of most American psychiatrists. In fact the manic phase is far less common than the depressive phase; the latter most often occurs alone, and with no manic phase either preceding or succeeding it. Whether the cases

[10] For details and theories regarding paranoia, see Mendel, Krafft-Ebbing (112), Kraepelin (108–109), Bleuler (17). The first two give the old and classical concept. Kraepelin substitutes for it the paraphrenias, Bleuler stands as the protogonist of the psycho-analytic school, with Meyer and Jellifee representing an American group emphasizing the dynamic temperamental school. Frankly, I believe most of the writing on paranoia has advanced the subject not one bit further than it was in 1880.

[11] See discussion in Vol. V of the American Psych. Ass'n. Trans., 19, 11.

with only a recurrent depression are of the same group as those with manic and depressed phases is a question.

The depressed phase may take the form of a simple but intense sadness, a state in which pleasure departs from all things and all relationships, in which there is a great and painful feeling of incapacity, with sluggish thought, lowered energy, and a tendency to regression and solitude. This type of depression, often of short duration, is especially common in the community, and is not regarded as "insane."[12]

From this simple depression, which to the causal observer is merely a prolonged "blues," to the cases where there are accusations against the self of all kinds, marked depressive and hypochondriacal delusion, where the patient has to be forced to eat or dress or sleep, where desperate suicidal attempts are made is a long series of mental states, with confusion and hallucinations, stupor, wild depressed excitement, many of which are difficult, if not impossible to separate from dementia praecox.

The excited or manic phase may be a mere exalted state, with talkativeness, increased activity, restlessness, increased sex desire, and often at impractical zeal and idealism which brings the patient into all kinds of trouble, because with this *increase* in activity and effort is a corresponding *decrease* in judgment and inhibition, which brings the individual into conflict with others or makes him their prey. Especially manifest is an increased feeling of power and importance, and egoism which reaches ludicrous heights. This simple manic state, often called hypomania, may go on to wild excitement and restlessness, destructiveness, flight of ideas, and all kinds of extravagant delusions leading to wild and often terrible acts. To see a patient shift perhaps overnight from a melancholia, with lowered ego feeling, great unhappiness, immobility and mutism; refusing to eat and wearing the appearance of the most complete dejection; to exaltation, with heightened ego feeling, happiness, greatly increased activity, talking continually, gorging himself with food, and appearing like Joy gone mad is to believe that there is an inner switch which swings from the negative to the positive pole of feeling through the action of some diabolical inner agency.[13]

[12] I venture to state, that if the mild community cases were included manic depressive insanity would easily be the most common mental disease.

[13] I exclude from discussion here the mixed phases of manic-depressive.

Whatever studies have been made upon brain and body organs postmortem and during life on the secretions, blood, spinal fluid and urine, throw no light on the essential pathology or the cause of manic depressive insanity. Even in the abrupt change that takes place from the depression to the manic phase nothing is found that corresponds at all to the mental cataclysm. At the present time all psychiatry can do is to class this disease with the functional psychoses, a disease without an organic basis. That such a conception leaves things in the air is not denying that it has a great convenience.

Thus this disease is utterly unknown, as to origin, aside from its familial relationships. It is at present more linked up with innate temperament than to any other factor, implying that it is largely congenital in origin. Certainly one sees it arise in people of emotionally unstable nature, quick to tears, and quick to exuberance, but just as certainly it arises in those whose life has been characterized by steady purpose and steady temperament. Moreover, the affairs of life do not seem to breed it—the patient may, when well, pass through the most harrowing experiences—lose money, be humiliated by ruin, be afflicted by disaster of one kind or another—and remain well. Then without any definite cause he passes into a deep depression. It seems likely that the mood changes are organically conditioned, due to some inner mechanism of a chemico-endocrinal visceral nature, which in its swerve from normal brings about the mental disease. Elsewhere in describing the mood change which I call anhedonia, I have spoken of the "energy feeling" as basically involved, and I believe this to be true of manic depressive insanity.[14]

Do manic depressive and dementia praecox change into one another? We shall see that in their hereditary relationship are facts suggestive of this possibility. Certainly it is very difficult at times to distinguish between the two diseases, and two groups of psychiatrists will often disagree about cases, and are without a fundamental diagnostic criterion, a definite objective sign. The agitated depression of manic depressive psychosis is difficult to separate from the depressed manifestations of dementia praecox; the stupor of manic depressive is hard to distinguish from the catatonic aspects of praecox, and it is nowadays asserted, and with reason, that there is a true catatonic phase in certain cases of manic depres-

[14] Anhedonia (143).

sive. I have seen, several cases of simple depression, that years later were followed by a psychosis of chronic type of typical catatonic character. At the borders these diseases meet, but typically they are quite distinct.

The involution psychoses. In the involution period, which follows the menopause in women, and starts in the late 40's and early 50's in men, there are two types of mental disease that occur, and which will appear in the family studies of the 2nd part. The first is called involution melancholia and was described by Kraepelin, and later by Dreyfus, Kirby, Bleuler Adolf Meyer, and others.

In the main this disease resembles the agitated depression of manic depressive insanity, and in the opinion of many writers belongs to the manic depressive group. There is a great depression associated with great fear (or perhaps the order of things should be reversed, there is fear associated with depression), there occur hypochondriacal delusions, and especially typical are the so-called nihilistic delusions which are not common in the depressed phases of manic depressive. One sees in the asylums many marked cases of this character, anxious, restless, wringing the hands and moaning, afraid to eat or sleep, asking piteously, "Why do I feel this way? Why is everything changed? Why am I so different? I have no bowels, no heart, no mind, all is gone, etc., etc." In private practice one sees the milder cases, with no pep, "all in," "no good any more, bound for the poor-house, past my day" people who are piteously convinced that the end of their career is come, that nothing but disaster awaits them. In the hospital about 50 to 60 per cent recover, in private practice recovery is more frequent.

The main psychological elements in this disease certainly bear a definite relationship to the time of life. It is in the involution period that death commences to figure largely in the thoughts, that the toll of friends and relatives increases rapidly. Further, there is a slackening of power first in the physical field, more especially in the sexual life, and also in the mental. Originality, energy, and initiative fail, there comes the realization that purposes cannot be fulfilled, and an intense questioning hypochondriacism results, which is present to some degree in all middle age, but which becomes exaggerated into a disease in these patients. The fear of the poor-house, of bodily disease, of death, of punishment for sins, find easy explanation in the very nature of the time of life. Some German

psychiatrist, and I regret that I have lost the reference, has with unusual insight declared all melancholia and especially the involution type, to be the disappearance of the "Life Lie"—the stripping away of the illusions by which the grim realities are camouflaged, the sudden realization of the fate that awaits man.[15] Though involution melancholia bears a resemblance to manic depressive in some aspects, it differs in others, especially in the fact that its symptoms can be more readily understood as the reaction to a life situation.

Involution psychoses, leading mainly to dementia. If involution melancholia in a general way resembles the depressed phase of manic depressive insanity (it is significant that there is no manic phase to involution melancholia) so there are diseases in the involution period which in a general way resemble dementia praecox. I do not wish in this volume to enter into any classification problem but I have at times cited certain cases of involution disease to psychiatrists without giving the age of onset and have invariably received the diagnosis "dementia praecox." The types vary about as dementia praecox does, there being cases of slow progression, with markedly paranoiac phases, and cases of rapid deterioration, which except for the age of onset, do not differ in gross ways from dementia praecox. We find the same apathy in some, the same fantastic and queerly incongruous hallucinations and delusions, in others catatonic phases are common, and so are the curled up bench types.[16]

Senile dementia. Roughly speaking, the senium is the period of life from 60 on and a large percentage of admissions to state hospitals take place after that time of life. The main types usually spoken of in this period of life are the organic psychoses, i.e., those following cerebral arterio-sclerosis, or associated with heart, kidney and arterial disease. These, I have already discussed in chapter III. The other group is usually discussed as senile dementia, by which is ordinarily understood a group of conditions occurring in the senium, not associated with any arterio-sclerotic condition. These senile diseases may be described as:

[15] All the pessimistic literature depends largely upon the disappearance of the "life lies." Only the strong of spirit can look upon life as it is and retain courage and ardor.

See for a wonderful discussion of melancholic trends Wm. James (91) magnificent "Varieties of Religious Experiences."

[16] These are known also as presenile dementia.

1. Senile deterioration, in which there is merely an accentuation of the memory loss and mental recession of normal old age;

2. Senile melancholia, which is practically the same as the depressed phase of manic depressive insanity except as it is associated with the mental deterioration of old age, and

3. Senile dementia proper, in which delusions, hallucinations, conduct disorder of all kinds, together with dementia appear. In some respects this strongly resembles dementia praecox.

4. Senile paranoia, in which delusions of persecution figure largely. In order to make clear the position taken in this book I quote from a former publication.[17]

Normally the senium carries on mental and physical changes that have begun after the earlier involution period. There is present physical enfeeblement and lowered capacity, and there is also present mental enfeeblement and lowered capacity. Also, because of the changed conditions of life, the retirement from activity and the dependence on others, there is a change in the mood and the social attitude, which may be summed up as querulous. In certain fortunate individuals, these changes play but little part; in others, the enfeeblement, etc., is quite profound. This is senile deterioration and, in my belief, ought sharply to be distinguished from the senile psychoses as having a relation to normality. Now this senile deterioration is part of the general picture presented by practically every patient with a senile psychosis, but it is not part of the psychosis. It is part of the normal mentality of the senium. Yet, as it often obscures a paranoia or a manic state, a catatonic outburst, or a dementia praecox occurring first at the senium, so also it obscures the psychiatrist's conception of senile diseases. The senile deterioration ought not to influence the diagnosis. Thus, for example, its co-existence with dementia praecox is not only possible but in my opinion very common and ought not to confuse the psychiatrist.

There are, furthermore, very good reasons why the senium (and the involution period) should be the period of the first appearance of psychoses. The very nature of conditions, both internal and external, both in the individual and in the environment, makes quite easy three mental attitudes. The first, a self-deprecation of an intense kind and apt to be associated with somatic delusions; the second, a keen apprehension and agitation; and the third, a hostility to, and a suspicious feeling directed against, the environment. The first, the self-deprecation and the falling physical and mental powers bring about in the predisposed individual a hypochondriacism that may, by a figure of speech, be said to extend to the soul as well as to the body, and thus somatic and spiritual delusions arise. The second, the apprehension and agitation, may be related to the fear of death. Death for the old is not a theoretical

[17] I am aware that this is a rough division, not nearly so elaborate as that of the American Psychiatric Association.

ending of life as it is for the young. It is the intensely dreaded, because very near, destroyer. The normal old man adjusts himself and his mood to his impending fate, though here too there is a quiet melancholy which sharply differentiates the old from the young. The abnormal old man finds the situation too difficult to bear and we have the involution and senile melancholia marked by intense agitation and apprehension. The third, the paranoid attitude, is explainable by the fact that at the senium the social environment does not become hostile in that the old are often regarded, perhaps unconsciously, as in the way. Their places are taken by the younger, they are swept out of the stream of activity willy nilly. So even in the normal senium (except in those rarely serene souls who reach an elevated resignation) there is an undercurrent of bitterness, a sense of injustice and ingratitude. In the person by nature suspicious, seclusive and hostile, delusions arise which are no different than the delusions of any other period except that they have been brought out and colored by the senium. So too, referring back to the hypochondriacism (and apprehension) it is not essentially different from that found in earlier life. It is perhaps more intense and more logically based because, on the whole, the senile dementia patient is a more resistant individual than the patient with dementia praecox.

It seems probable that in all the periods of life there occur three main types of mental disease. The one is a paranoiac type, a disease with hostility, suspicion, a deluded interpretation of the life around it and a gross egoistic, over-evaluation of the self. The second is a dementing disease, whose prototype is dementia praecox, but which occurs in modified form in the involution and the senile periods. The third is a disease marked mainly by a depressed mood, with lowered energy, absent interest and delusions of a depressive, apprehensive nature, occasionally associated with excitement and exaltation. The main disease of this type is manic depressive insanity but the involution and senile melancholia are related diseases, perhaps the same disease colored by the emotional and mental reactions of these periods of life.

It might thus be stated—that these three types of mental disease may occur at any period of life, in youth, maturity, involution or senium. Whatever is their cause, the less resistant individuals develop these conditions early, others more resistant hang out until the changes of the involution, and still more resistant individuals develop them late in life. That this is more than a hypothesis I do not claim, indeed it can only be called a tenuous hypothesis at the best. But it fits in fairly well with facts to be cited in the next part of this book, facts which deal with the heredity of these psychoses.

PART II

INTRODUCTION

One who has been vigorous in his criticism of others must apologize for repeating errors he has pointed out in their work. It will be noticed that in the histories of the 130 families who are studied in this part of the book that the words insane, feeble-minded, epileptic without further qualification, occur time and again in the family histories of the patients. There are two facts that mitigate, so to speak, the offence against accuracy and science in such statements.

In the first place no effort has been made to draw any conclusions from these statements. The only conclusions made relate to the patients who have been in the Taunton State Hospital, concerning whom there are records, and who have been examined either by myself or by predecessors trained in psychiatry. I have made no attempt to solve the ultimate problems of the transmission of mental diseases on any data to be obtained by family history, by social workers, or field workers. Whatever problems this part of the book attacks are definite clinical problems, and can be solved by the method used.

In the second place, the family study finally undertaken was a small part of the plan projected. It was hoped at the time (1915–1917) when the work was commenced to study all relatives of the patients at the hospital, to study the normal ancestors and descendants in so far as possible. Unfortunately no finances were forthcoming, and neither state, private or public agency could be enlisted in the work, so it was necessary to do what could be done by one person, whose routine duties as clinical director and pathologist were heavy and whose researches had to be done at odd moments and under discouraging circumstances.[1]

[1] To my colleagues at the Taunton State Hospital, and especially to Dr. Arthur V. Goss, then the superintendent of the hospital, I owe much for their interest and help in this work. To Dr. Charles Lipschutz then a medical student, to Miss Marian Sweet, and Mr. Ross J. McCann I am indebted for help in clerical work and in the drawing of diagrams. This help never received any financial returns; it was given freely and gladly, and is here acknowledged in the same spirit. Especially, and gratefully, do I give to my predecessor as pathologist to the hospital, Dr. Charles G. McGaffin, the great credit due him for his start in the work here carried out.

The Taunton, Massachusetts, State Hospital, was founded in 1854, and deals with the population of southeastern Massachusetts. On the one hand it receives mental cases from the Cape Cod District, from one of the earliest settled parts of the United States, where there still live the descendants of the Pilgrims, where conditions of life are rather fixed and settled, where conservatism in act and thought is held as holy, but from which district sons and daughters have come who have helped people and direct much of the rest of the country.

On the other hand it receives patients from the mill towns of New Bedford, Fall River, Brockton, and Taunton, where there is as large a proportion of foreigners and recent immigrants as anywhere in the United States. The hospital population is thus made up of the old and the new stocks of America, and a host of problems are presented to the interested observer. Of these problems I have here chosen the relation of the psychoses to each other as they appear in parents and descendants, and in brothers and sisters.

There is an additional statement to make to explain certain changes that appear in this account from that as made in my original publication. At that time my psychiatric experience had been entirely institutional. Since then I have added an extensive experience in the out-patient department of the Boston psychopathic Hospital and in private psychiatric practice. Out-patient and practice psychiatry bring one in contact with phases of mental disease that the institutional psychiatrist never meets, and one learns much which has never reached the literature. One sees all kinds of mental disease arising in the descendants of individuals apparently normal and in whose family history there is little abnormality, unless one raises into the neuropathic class qualities altogether too common to be given, off-hand, such sinister significance, viz., headache, irritability, crankiness, sex offence, family discord, neurasthenia. One sees that there is not that sharp line between "normality" and "insanity" which one imagines in custodial psychiatry where one deals with end products, that in the "normal" person during illness and fatigue, under the stresses and strains of adversity there arise transitory paranoias and depressions. Even to see the great changes which take place in the descendants of immigrants in one generation, changes affecting character, personality, and disease, is to realize that he who deals with man and lightly dismisses the environment in its great rôle does not know life or mental disease.

Normal and "Insane" Heredity Contrasted

Three important and much quoted pieces of work have been carried out to determine whether or not it is justifiable to lay so much stress on the heredity of insane persons as causative of their psychoses or as bearing any intimate relation to it by using as a control the insane and neurotic heredity of the non-insane. I am first taking the analysis of the papers of Koller and Diem as made by Von Jauregg (94), slightly altering the figures to make them approach round numbers.

Koller and Diem working in the same district some fifteen years apart in time contrasted the heredity of the insane and normal as elicited by the method of family history. In both sets of studies vitiating factors arise. The first is, that considered among the normal are the eccentric, psychopathic, and peculiar as well as the hospital patients of non-insane[2] kind; and the second, that in the majority of cases the normal were under thirty, whereas the insane ages ranged much higher. The first error obviously raises the psychopathic heredity of the normal. The second ignores the great fact that the incidence of "insanity" increases with the years so that of any group of young people a certain number, and especially of the psychopathic and peculiar young people, some certainly will later "go insane." Therefore, while the statistics of the insane stand by themselves as pure, those of the normal are tainted by the figures that pertain to some potential insane persons. While this obviously raises the psychopathic heredity of the normal other vitiating factors are the ones so frequently mentioned in this book. The term insane and the term nervous are given too broad a scope and lose their value because of this. Further, when speaking of the insane they do not separate them into groups and so we have amongst the insane such individuals as are suffering from arteriosclerotic dementia or general paresis in which it is on the whole unlikely that heredity plays an important part. Nevertheless the facts presented by Koller and Diem have some value and it is for this reason they are cited here.

In the following tabulation the term "total taint" means the insane and neuropathic heredity of all grades of ancestors. I have sub-

[2] I use the terms insane and insanity in this account of the work of Koller and Diem because it is their habit and I cannot go behind the terms to discover their meaning.

stituted the term "parental taint" for the term direct taint as used by Koller and Diem, and the term "collateral taint" to mean that which is carried through an uncle or an aunt.

1. Total taint of insane compared to that of sane is as

1.3 : 1, Koller.
1.1 : 1, Diem.

This shows only a small preponderance of total taint in the insane and shows its real value, which is very little.

2. Mental disease as a taint in heredity of insane as against that in the sane is as

2 : 1, Koller.
$4\frac{1}{2}$: 1, Diem.

This shows a striking preponderance of insane taint in the heredity of the insane and also a striking difference in the percentage of results of the two workers.

3. Parental taint of mental disease in the heredity of the insane as against the same in the heredity of the sane is as

3.7 : 1, Koller.
8.3 : 1, Diem.

The preponderance of the insane heredity becomes more striking as the direct line is approached, and the two workers while agreeing in the direction of the current differ very markedly in the estimate of its force.

4. The total taint of uncles and aunts in the insane as against the same in the heredity of the sane is as

1.14 : 1, Koller.
0.37 : 1, Diem.

Here is a striking divergence of statistical results.

The one author finds the insane slightly more tainted through collateral ancestors, while the other finds the sane are about three times as heavily tainted through collateral ancestors.

5. The total taint of insane uncles and aunts in the heredity of the insane as against the same in the heredity of the sane is as

1.05 : 1, Koller.
0.87 : 1, Diem.

The results are nearly equal for the sane and the insane according to both workers.

6. The total taint of grandparents in the heredity of the insane as against the same in the heredity of the sane is as

0.5 : 1, Koller.
0.33 : 1, Diem.

Here we have a striking reversal of expectations, for the sane are far more tainted than the insane.

7. The total taint of insane grandparents in the heredity of the insane as against the same in the heredity of the sane is as

1.66 : 1, Koller.
3.4 : 1, Diem.

Thus, despite the fact that the total taint is greater in the sane, insane heredity through grandparents is far more common in the insane. Total taint thus becomes of no importance.

8. Nervous disease as a taint in the heredity of the insane is to the same in the heredity of the sane as

6 : 1, Koller.
.28 : 1, Diem.

This needs no comment except that it reinforces the attitude I have consistently assumed.

9. Apoplexy as a taint in the heredity of the insane as against the same in the heredity of the sane is as

0.5 : 1, Koller.
0.25 : 1, Diem.

This fact certainly does not make apoplexy an important psychopathic character. In fact it gives it a "normal" significance.

10. Senile dementia in the heredity of the insane is to the same in the heredity of the sane as

1 : 1, Koller.
0.14 : 1, Diem.

This is the most striking divergence in the statistical results of the two workers, and shows the uselessness of statistical study if insufficient cases are considered.

11. Character anomalies in the parents of the insane are to the same in the parents of the sane as

3 : 1, Koller.
$2\frac{1}{3}$: 1, Diem.

There is agreement here. The insane have parents with character anomalies from two to three times as frequently as do the sane. It is, however, probable that sane people would not be quite frank about the character anomalies of their parents.

12. Character anomalies in the other relatives of the insane are to character anomalies in the other relatives of the sane as

1 : 1, Koller.
37 : 1, Diem.

This shows, if anything, that the indirect relatives are more heavily afflicted in the sane than in the insane.

13. Alcoholism in the parents of the insane as against the same in the heredity of the sane is as

2 : 1, Koller.
$1\frac{1}{2}$: 1, Diem.

14. Alcoholism in other relatives of the insane as against the same in the history of the sane is as

0.66 : 1, Koller.
0.6 : 1, Diem.

Here the two workers are in accord that alcoholism in the parents is more frequent in insane heredity, whereas alcoholism in other relatives is more common in the sane.

It thus appears that the insane are slightly more tainted, if all degrees of taint in all relatives (excepting children) are considered. They are far more tainted by "insanity" and more especially by insanity in the parents and through direct inheritance by character anomalies, and somewhat more heavily through direct inheritance by alcohol. On the other hand, the sane have more nervous diseases, apoplexy, senile dementia in their heredity and through their collateral ancestors. In general they are more heavily tainted through grandparents and collateral ancestors than are the insane.

It thus stands out that the important factors to be gathered in the heredity of any person in so far as mental diseases are concerned is the existence of mental disease in his direct relatives, in his parents, in his brothers and sisters, and his grandparents, rather than in his uncles and aunts. Furthermore, as I have insisted in the previous pages the type of mental disease is important and must be ascertained to make the statistics of any definite value. Alcoholism in the *parents* probably occurs more commonly in the ancestors of the insane

than in the ancestors of the sane, yet in itself cannot be given a causative value. It is to be weighed as a factor in each case by itself.

Jolly (99) studying two-hundred normal and two-hundred mentally sick comes to the following conclusions. The total hereditary taint in the old sense is not much different in the sane than in the insane, 46.5 per cent former, as against 64.5 per cent latter. The mentally sick, however, show a great deal more of mental disease in the ancestry than do the normal, while on the other hand organic nervous diseases, apoplexy, and the functional nervous diseases are more common in the ancestry and relatives of the normal than those of the abnormal. Quoting Hoffman:

> It follows that mental disease, character anomaly, and the type of alcoholism which is related to character anomaly, further also suicide, are real hereditary taint factors (Belastungs moment) of the mentally sick, but not the functional and organic nervous diseases, or apoplexy.

Thus Jolly corroborates the work of Koller and Diem, and thus it becomes ridiculous to include as factors in producing the psychoses those diseases and disease conditions which are more common in the ancestry and relatives of the sane than in those of the "insane."

Wagner Von Jauregg quoting the figures of Koller and Diem makes some observations that have at least the merit of striking originality.

> Disposition or predisposition is a concept which has a complement and that complement is immunity. Thus, taint through uncles, aunts, and grandparents may be considered as an immunizing factor rather than the reverse.

This last is obviously an overstatement so far as insanity in these ancestors is concerned, according to Koller's and Diem's figures. Moreover, it is impossible, at least for me, to picture any process by which any disease in an uncle or aunt confers immunity on a nephew or niece, whereas disease in the direct line through a parent confers no immunity.

Marriage Rate of Insane

A vitally important matter in the discussion of heredity and hereditary diseases in the "insane" or any other group is the marriage rate of the individuals concerned. Do the psychoses interfere with the marriage rate, or rather does the condition underlying a psychosis—alcoholism, syphilis, psychopathic constitution—interfere with marriage? Do the male and female insane marry in equal ratio: This,

of course, bears upon the transmission of "insanity" through male and female ancestors, a problem which has received much attention.

I have, therefore, taken from the Taunton State Hospital records several hundred cases, male and female, of each of the following groups: Alcoholic psychoses, general paresis, dementia praecox, and senile psychoses. It was easily possible to obtain 500 consecutive males in each of these groups, and likewise 500 females, dementia praecox and seniles. In alcoholism 450, and in general paresis 250 female cases were all that were available.

	ALCOHOLIC		PARESIS		DEMENTIA PRAECOX		SENILE PSYCHOSES	
	Male	Female	Male	Female	Male	Female	Male	Female
Single	43.2	14.9	22.0	13.6	72.0	48.4	84.0	14.6
Ratio	3 m. to 1 fem.		1.5 m. to 1 fem.		3 m. to 2 fem.		1 m. to 1.6 fem.	
Married	46.2	60.0	66.8	68.9	23.4	42.2	46.0	22.4
Ratio	3 m. to 4 fem.		Nearly equal		1 m. to 2 fem.		2 m. to 1 fem.	
Widowed	9.2	20.7	9.9	15.7	1.8	6.2	43.8	61.8
Ratio	1 m. to 2 fem.		1 m. to 1.5 fem.		1 m. to 3.5 fem.		2 m. to 3 fem.	
Divorced	1.4	4.4	1.3	2.5	2.4	2.8	0.8	1.0
Ratio	1 m. to 3 fem.		1 m. to 2 fem.		Nearly equal		Nearly equal	

If now the psychoses are brought into closer comparison as regards the totals of conjugal state, that is, whether or not the individual has married at any time past or present, certain facts stand out.

	ALCOHOLISM	PARESIS	DEMENTIA PRAECOX	SENILE
	per cent	*per cent*	*per cent*	*per cent*
Male:				
Married	60	80	30	85
Non-married	40	20	70	15
Female:				
Married	85	85	50	85
Non-married	15	15	50	15

Finally, I have brought together these figures in so far as the single largest group of each psychosis, male patients, is concerned with a similar group of the total population and compared the marriage

ratios between these insane groups and the corresponding total population group.

		per cent M.W.D.
Male alcoholics		
Largest group of 500, 35–49 years	238	55.9
Total population, males, between 35 and 44 years		83.1
Male paretics		
Largest group of 500, 35–49 years	293	78.0
Total population, males, between 35 and 44 years		83.1
Male dementia praecox		
Largest group of 500, 20–34 years	309	18.0
Total population, males, between 20 and 34 years		49.0
Male senile psychoses		
Largest group of 500, 60 years and over	459	83.2
Total population, males, between 60 years and over		93.5

In the above groups the alcoholics and paretics are compared with an age group in a total population whose outer limit is five years greater. This accentuates the difference shown to exist, because older age groups, other things being equal, show a greater percentage of marriage than younger age groups.

Summarizing the results, we find first, that the males in the alcoholic, paretic, and dementia praecox groups marry less than do the females. In the seniles, though the percentage of married men is greater, the totals of those who have entered conjugal relations at one time or another are about equal. Looking somewhat closer, it is found that in paresis there is only a slight difference in favor of the female. In alcoholics there is a very decided difference in favor of the female, and in dementia praecox this difference is still further increased. Thus, if these groups may be held to constitute a menace by virtue of their ability to transmit the psychotic taint to another generation, the female of the species, to use a well-known phrase, is more dangerous than the male.

It will be seen that in the four diseases the females marry in about the same ratio, except in the case of dementia praecox where the falling off is considerable. In the males there is much more decided irregularity in marriage ratios. Whereas the seniles and the general paretics marry but slightly less than do the same age groups in the total population, the alcoholics show a decided falling off as compared with the total population, while the male dementia praecox has an exceedingly low marriage rate. *This is, whatever is back of dementia praecox, it operates against self-perpetuation.* Something of the same

internal mechanism is seen in the case of alcoholic psychoses. This mechanism operates very little, if at all, in the case of paresis and the senile psychoses. One might conclude that if there is an inborn defect in these diseases it is by far greatest in dementia praecox, is next in alcoholic insanity, and least of all in syphilis and the senile psychoses. And in a general way this will be found to correspond with the belief of the average psychiatrist. For most of us, despite some authorities, paresis is held to be wholly an exogenous disease caused by the Spirochaeta pallida working on a relatively intact organism. If Koller's and Diem's figures form any criterion, senile psychoses represent conditions that are largely exogenous in their origin. Alcoholic insanity in most clinics is held to be the result of exogenous and endogenous factors, while dementia praecox is believed to be largely of endogenous origin.

Should the figures thus obtained at Taunton be substanitated by workers in other centers, then certain facts concerning marriage itself stand out as important. It is the belief of statisticians whose work is concerned with the total population that marriage exercises a beneficent influence upon longevity, in that married men live longer than single and that widowers and divorced men show a higher death rate than the married; whereas, while the same influences in general work on the female population they work to a lesser degree, in that married women show only a moderate degree of increased longevity as compared with spinsters. While it may be true that the marital relationship is conductive to greater length of life, there is another factor at work which these statistics on the marriage rate of the insane show. *That factor is that the more normal of the population tend to marry, whereas the more abnormal of the population tend to remain single. This, of course, is only a rule, broadly speaking, and does not apply with the same force to women as it does to men because the male, despite the witty George Bernard Shaw and others, is the active agent in selection and proposal so that a greater number of normal women remain unmarried and a greater number of abnormal women become married.* (Which perhaps is an argument for feminism but is not so adduced). With this greater abnormality of the unmarried comes a higher death rate, for it is well known that mental peculiarities do not stand alone and are associated with greater vulnerability to disease.[3]

[3] Those who are interested in the relation of sex to mental disease will find a discussion of this matter in the works of Kraepelin, who believes that the

We are shown that marriage acts as a barrier to the propagation of the abnormal in so far as this is connected with endogenous factors. It is not a barrier against certain of the exogenous race poisons, such as, for example, syphilis, at least in the form which leads to paresis. We need to strengthen the barrier against the endogenous diseases, for example, dementia praecox, but not nearly so much as we need to strengthen it against the exogenous, as, for example, syphilis.

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influence of the father is greater than the mother, Frankhauser (61) who disagrees with this, Schuppius (174), Wittermann (218), Krueger (116), and Luther (125). Hoffman (83) summarizes the statements of these authors as follows: "This review of the rather incomplete researches shows that no simple or definte result of the relation of the sex to heredity in the psychoses can be stated. . . . . There seem, however, to be a group of investigators who unite in the belief that the female sex on the whole inclines more to mental disease phenomena than the male."

CHAPTER VIII

Statistics of the Taunton Cases

There had been at the time that this part of the work was completed, in January, 1916, 22,300 admissions to the Taunton State Hospital since its founding in 1854. From a rough calculation made by analyzing 3000 cases taken at various points in the history of the hospital, it seems that about 16,000 persons are represented in the 22,300 commitments.[1] Of the 1300 people in the hospital at that time, roughly 10 per cent were related to one another. Of the patients who had been in the hospital from 1854 to 1916 there were 1547 who were related to one another and these represented 664 families.

1 four generation family.
23 three generation families.
- 7 cases three generations represented.
- 16 cases first and third generations represented.

333 two generation families.
- 189 direct relationship (father and mother, descendant).
- 112 collateral relationship (uncle or aunt, descendant).
- 32 mixed (direct and collateral), father or mother, and uncle or aunt, descendant.

307 one generation families divided as follows:
- 247 sibling families (brothers and sisters).
- 51 collateral families (cousins).
- 32 mixed (siblings and collaterals), brothers or sisters and cousins.

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664 total.

Females	808
Males	739
Total	1547
	times
Father-daughter relationship occurred	59
Father-son relationship occurred	55
Mother-daughter relationship occurred	80
Mother-son relationship occurred	56
Uncle-niece relationship occurred	37
Uncle-nephew relationship occurred	41

[1] That is many patients have been committed more than once.

Aunt-niece relationship occurred..... 42
Aunt-nephew relationship occurred..... 43
Cousinship relationship occurred..... 73
Brothers, alone, groups..... 57
Sisters, alone, groups..... 80
Brother-sister groups..... 110
Husband-wife groups..... 36

Mother-daughter relationship greater than father-daughter.
Mother-son relationship about equal to father-son.
Aunt-nephew relationship greater than uncle-nephew.
Aunt-niece relationship greater than uncle-niece.
Sister groups greater than brother groups.
Total females greater than total males.

This compares very closely with Mott's (137) figures from London County Asylum. Since London County presents a social picture very different from that represented by the district which Taunton State Hospital drains, it may be inferred that similar situations prevail throughout the world.

Analysis of 3118 related cases, instances of two of a family insane
Mott

	PAIRS	CASES
Mother and daughter	137	314
Mother and son	96	192
Father and daughter	103	206
Father and son	78	156
Brothers and sisters	212	424
Two sisters	211	422
Two brothers	140	280
Husband and wife	69	138
Offspring and grandparents	24	48
Other relationships, collaterals, etc	186	372
Total	1276	2552

142 instances of 3 of a family insane..... 426
24 instances of 4 of a family insane..... 96
5 instances of 5 of a family insane..... 25
2 instances of 6 of a family insane..... 12
1 instance of 7 of a family insane..... 7

Total..... 3118

Total—3118 cases made up from 1450 families.

A striking series of facts appear under close analysis of my figures. The mother-son relationship is much less frequent than mother-daughter (as 55 is to 80), but the father-son relationship is only slightly less common than the father-daughter (as 55 is to 59) and represents a difference more likely to be accidental. One might feel safe in stating that insane mothers tend to have insane daughters more frequently than insane sons, whereas no direct statement is warranted by the father and descendant figures. In this connection the statement of Tigges is of importance. He states that in direct maternal heredity, daughters everywhere are more frequently found to be insane than are daughters through direct paternal heredity. But collateral ancestors, uncles and aunts and descendants, by my figures run closely together—37 and 41 for uncle groups; 41 and 43 for aunt groups. Sisters alone greatly outnumber brothers alone, whereas brother-sister groups are about twice as common as brother alone and one and three-eighths as common as sisters alone.

Is this predominance of females to be ascribed to the condition that Mott finds responsible?

> The physiology and emergencies connected with reproduction—the menstrual periods, child-bearing and the cessation of the period of reproduction, the climacterion. Moreover there is a more unstable equilibrium in women. I would also add as an important and perhaps the one cause in many instances, the enforced suppression by modern social conditions of the reproductive functions and the maternal instinct of women of an emotional temperament and mental instability.

Now no one can deny that these factors play a part, *but if insane women transmit their mental peculiarities to their female children more than they do to their male, then the greater marriage rate amongst insane women may decidedly play a part in determining the preponderance of insane women.* Furthermore, men migrate more than women, and so in any given asylum district the female descendants of insane ancestors would be more apt to appear in the asylum than the male descendants even if given equal rate of incidence. That is to say, *a larger part of these men would end in jails or in insane hospitals in districts remote from their former home, etc.* I do not believe that the great function of reproduction in women is an exciting factor of more importance in determining insanity than is the greater social stress of men's lives.

"Anticipation or Antedating." This term is used to describe the earlier appearance of mental disease in the younger as compared with the older generation. This phenomenon has been given great prominence by Mott and his co-workers and he regards it as an effort of nature to get rid of the disease by crystallizing it in a few descendants and making them more easily vulnerable or unfitted to propagate by being brought early to asylums. There are thus to Mott's definition of anticipation, first, *a crystallization* of the insane elements leaving other descendants free from disease, and second, *earlier onset* of the psychosis in the affected members. I here subjoin Mott's figures on "Anticipation or Antedating," the first table referring to this phenomenon as it relates to direct heredity, the second as it relates to collateral heredity.

Statistical data relating to inheritance and insanity, especially in relation to anticipation

From an investigation of the age at the time of first attack in 508 pairs of parent and offspring (from the records of 464 insane parents of 500 insane offspring), the following table has been compiled. The figures denote the percentage of cases whose first attack occurred within the given age-periods.

AGE-PERIODS	FATHER	OFFSPRING	MOTHER	OFFSPRING	
Under 20 years	1.4	26.2	0.6	27.8	
20–24 years	0.4	18.0	3.4	15.7	*
25–29 years	1.4	18.0	4.4	18.2	*
30–34 years	9.6	13.0	7.8	13.4	
35–39 years	11.5	7.3	9.2	10.0	
40–44 years	9.2	6.4	10.3	5.8	
45–49 years	14.3	6.0	12.0	3.7	†
50–54 years	17.5	0.9	12.3	2.4	†
55–59 years	13.8	3.7	14.0	1.7	
60–64 years	10.1	—	11.6	1.3	
65–69 years	5.0	—	8.8	—	
70–74 years	4.6	0.4	3.1	—	
75–79 years	0.4	—	1.3	—	
80 years	0.4	—	0.6	—	

AGE-PERIODS	COLLATERAL ONLY		COLLATERAL AND DIRECT		
	Uncle or aunt	Niece or nephew	Uncle or aunt	Niece or nephew	
Under 20 years	5.2	20.7	5.2	25.5	
20–24 years	3.1	19.2	3.4	17.7	*
25–29 years	6.2	18.6	7.8	19.0	*
30–34 years	12.9	17.1	14.3	15.1	
35–39 years	11.9	12.4	12.1	11.2	
40–44 years	11.3	5.7	10.5	4.3	
45–49 years	12.4	2.1	12.1	2.6	†
50–54 years	14.5	2.1	12.1	1.7	†
55–59 years	7.7	1.5	8.6	2.1	
60–64 years	8.8	—	8.2	—	
65–69 years	1.5	0.5	1.7	0.4	
70–74 years	1.0	—	1.3	—	
75–79 years	3.1	—	2.6	—	
80 years	—	—	—	—	

* Adolescence.

† Involution period.

It will be seen that according to Mott there is a tendency for the first attack to occur earlier in the descendants of both direct and collateral insane ancestors. I have arranged my cases in which such data were definite and those of Jolly, Albrecht, and Rosanoff somewhat differently in order to bring out the age-difference at onset rather than the age of onset. The following tables are self-explanatory.

FATHER AND DESCENDANTS (TAUNTON CASES)

A. Ancestor older than descendant at age of onset..... 67 cases

1. Difference of 25 years and over between onset of psychosis in father and descendant.

33 families—18 sons, 17 daughters

Ancestor between 30 and 40—none
Ancestor between 45 and 50— 7 cases, 27 years average difference
Ancestor between 50 and 60— 8 cases, 33 years average difference
Ancestor between 60 and 70—12 cases, 29 years average difference
Ancestor between 70 and 80— 2 cases, 32 years average difference
Ancestor between 80 and 90— 4 cases, 50 years average difference

2. Difference of 20 to 30 years between onset.

11 families—5 sons, 6 daughters

Ancestor between 30 and 40—1 case
Ancestor between 40 and 50—5 cases

Ancestor between 50 and 60—2 cases
Ancestor between 60 and 70—1 case
Ancestor between 70 and 80—2 cases

3. Difference of 15 to 20 years between onset.
7 families—5 sons, 5 daughters
Ancestor between 30 and 40—2 cases
Ancestor between 40 and 50—3 cases
Ancestor between 60 and 70—2 cases

4. Difference of 5 to 15 years between onset.
14 families—9 sons, 5 daughters
Ancestor between 20 and 30—1 case
Ancestor between 30 and 40—3 cases
Ancestor between 40 and 50—5 cases
Ancestor between 50 and 60—1 case
Ancestor between 60 and 70—2 cases
Ancestor between 70 and 80—2 cases

5. Difference of 0 to 5 years between onset.
2 families—1 son, 1 daughter
Ancestor between 40 and 50—1 case
Ancestor between 50 and 60—1 case

B. Descendant older than ancestor at age of onset..... 12 cases

1. Difference of 0 to 5 years between onset.
8 cases—4 sons, 4 daughters
Ancestor between 20 and 30—2 cases
Ancestor between 30 and 40—2 cases
Ancestor between 40 and 50—4 cases

2. Difference of 5 to 10 years between onset.
2 cases
Ancestor between 50 and 60—1 case
Ancestor between 60 and 70—1 case

3. Difference of 15 to 20 years between onset.
1 case—1 daughter
Ancestor between 20 and 30—1 case

4. Difference of 25 years and over between onset.
1 case—1 daughter, 30 years average difference

Father older than descendant is to the reverse as 67 is to 12. The greater number of cases fall in the groups where the ancester is much older, while the majority of the cases where descendant is older than ancestor at onset show only slight difference.

Mothers and Descendants

A. Ancestor older than descendant at age of onset..... 76 cases

1. Difference of 25 years and over between onset of psychosis in mother and descendant.

30 cases—21 daughters, 18 sons

Ancestor between 30 and 40— 3 cases, 30 years average difference
Ancestor between 40 and 50— 4 cases, 29 years average difference
Ancestor between 50 and 60— 8 cases, 30 years average difference
Ancestor between 60 and 70—10 cases, 34 years average difference
Ancestor between 70 and 80— 5 cases, 36 years average difference

2. Difference of 20 to 25 years between onset.

13 cases—10 sons, 9 daughters

Ancestor between 20 and 30—1 case
Ancestor between 30 and 40—1 case
Ancestor between 40 and 50—5 cases
Ancestor between 50 and 60—5 cases
Ancestor between 60 and 70—1 case
Ancestor between 70 and 80—1 case

3. Difference of 15 to 20 years between onset.

17 cases—11 daughters, 9 sons

Ancestor between 20 and 30—1 case
Ancestor between 30 and 40—2 cases
Ancestor between 40 and 50—7 cases
Ancestor between 50 and 60—4 cases
Ancestor between 60 and 70—2 cases
Ancestor between 70 and 80—1 case

4. Difference of 5 to 15 years between onset.

11 cases

Ancestor between 20 and 30—1 case
Ancestor between 30 and 40—3 cases
Ancestor between 40 and 50—3 cases
Ancestor between 50 and 60—3 cases
Ancestor between 60 and 70—1 case

5. Difference of 0 to 5 years between onset.

5 cases—5 sons, 1 daughter

Ancestor between 30 and 40—4 cases
Ancestor between 50 and 60—1 case

6. Where all members are about the same age.

5 families—13 members

B. Descendant older than ancestor at age of onset..... 11 cases

1. Difference of 0 to 5 years between onset.

3 cases—3 daughters

Ancestor between 20 and 30

2. Difference of 5 to 10 years between onset.

4 cases—4 daughters

Ancestor between 20 and 30—2 cases
Ancestor between 30 and 40—2 cases

3. Difference of 10 to 15 years between onset.

4 cases—1 son, 3 daughters

Ancestor between 20 and 30—2 cases
Ancestor around 40—2 cases

90 cases

Mother older than descendant is to the reverse as 76 is to 11. Same observation in majority of cases as in father and descendants.

Uncles and Descendants

A. Ancestor older than descendant at age of onset..... 39 cases

1. Difference of 25 years and over between onset of psychosis in uncle and descendant.

13 cases—9 nephews, 5 nieces

Ancestor between 40 and 50—4 cases, 27 years average difference
Ancestor between 50 and 60—5 cases, 29 years average difference
Ancestor between 60 and 70—3 cases, 38 years average difference
Ancestor between 70 and 80—1 case, 27 years average difference

2. Difference of *15* to *25* years between onset.

8 cases—5 nephews, 5 nieces

Ancestor between 30 and 40—3 cases
Ancestor between 40 and 50—3 cases
Ancestor between 70 and 80—1 case
Ancestor between 80 and 90—1 case

3. Difference of *10* to *15* years between onset.

11 cases—5 nephews, 6 nieces

Ancestor between 30 and 40—5 cases
Ancestor between 40 and 50—1 case
Ancestor between 50 and 60—5 cases

4. Difference of *0* to *5* years between onset.

7 cases—9 nephews, 1 niece

Ancestor between 20 and 30—3 cases
Ancestor between 30 and 40—1 case
Ancestor between 40 and 50—2 cases
Ancestor between 50 and 60—1 case

B. Descendant older than ancestor at age of onset.....5 cases

1. Difference of *0* to *5* years between onset.

3 cases—2 nephews, 1 niece

Ancestor between 20 and 30

2. Difference of 7 years between onset.

1 case—1 niece

Ancestor between 40 and 50

3. Difference of 13 years.

1 case—1 nephew

Ancestor between 20 and 30

Uncles older than descendants is to the reverse as 30 is to 5.

Aunts and Descendants

A. Ancestor older than descendant at age of onset..... 32 cases

1. Difference of 25 years and over between onset of psychosis in aunt and descendants.

16 cases—10 nephews, 6 nieces

Ancestor between 40 and 50—2 cases, 31 years average difference
Ancestor between 50 and 60—5 cases, 36 years average difference
Ancestor between 60 and 70—6 cases, 40 years average difference
Ancestor between 80 and 90—2 cases, 60 years average difference

2. Difference of *15* to *25* years between onset.

8 cases—4 nephews, 4 nieces

Ancestor between 30 and 50—4 cases
Ancestor between 50 and 60—2 cases
Ancestor between 60 and 70—1 case
Ancestor between 70 and 80—1 case

3. Difference of 10 to 15 years between onset.

6 cases

Ancestor between 20 and 30—1 case
Ancestor between 30 and 40—4 cases
Ancestor between 40 and 50—1 case

4. Difference of *0* to *5* years between onset.

2 cases

Ancestor between 50 and 60—1 case
Ancestor between 60 and 70—1 case

5. Non-pertinent cases, 2 cases.

B. Descendant older than ancestor at age of onset..... 13 cases

5 cases—3 nephews, 2 nieces

1. Difference of less than *5* years between onset.

Ancestor between 20 and 30—2 cases
Ancestor between 30 and 40—1 case
Ancestor between 40 and 50—2 cases

2. Difference of *5* to *10* years between onset.

4 cases—2 nephews, 2 nieces

Ancestor between 20 and 30—3 cases
Ancestor between 40 and 50—1 case

3. Difference of *15* to *25* years between onset.

4 cases

Ancestor between 20 and 30

Aunts older than descendants is to the reverse as 32 is to 13.

The following tables arranged from the cases cited by Rosanoff and Jolly will sufficiently corroborate the statistics of Mott and those of mine.

JOLLY TABLE

Ancestors older than descendant at time of onset

In 7 cases 25 years or more
In 7 cases 20–25 years
In 8 cases 15–20 years
In 3 cases 10–15 years
In 9 cases 0–10 years

ROSANOFF TABLE

Ancestors older than descendant at time of onset

In 11 cases 25 years or more
In 2 cases 20–25 years
In 8 cases 15–20 years
In 5 cases 10–15 years
In 2 cases 5–10 years
In 1 case no difference

In five cases the descendant was older than the ancestor. In two of these, however, the descendant had epilepsy for many years before the time of the onset of the psychosis, leaving only three cases in which the ancestor was younger than the descendant.

It may be stated further that all of Albrecht's cases show a greater age of ancestor as do practically all of Krueger and Luther. Here arises the question, "What do the cases show in which institutional insanity has occurred for more than two generations?" Mine are the only recorded cases and of these many are either in part in the indirect line or the middle generation has not appeared at the hospital. In the four-generation case there is moderate and fairly steady antedating; first generation, 48 years at time of onset; second generation, two individuals 28 and 40 years; third generation, 30 years; fourth generation, 24 years.

In the third generation cases the situation is not so clear because of the difficulties above mentioned. On the face of things there is moderate antedating visible here. Of 23 families, 16 show the phenomenon and seven show none or else a slight tendency in the opposite direction. Roughly speaking, only the cases with a senile psychosis in the ancestor show antedating to a marked degree. Cases with manic-depressive insanity or dementia praecox in the ancestor do not show antedating to anything like the same degree. The following table gives the figures of these three generation cases. The figure in parenthesis refers to the case number; the numeral to the age of onset:

(1) 47–35–23; (2) 64–42–23; (3) 70–71–50; (4) 53–17 (1st & 3d gen.), 28–17 (2d & 3d gen.); (5) 66–27–35; (6) 28–30–31; (7) 62–32–31; (8) 54–25–17; (9) 20–?–19; (10) 70–7–18; (11) 28–20–18; (12) 34–23–30; (13) 70–?–16; (14) 80–?–imp.; (15) 68–?–28; (16) 60–?–30; (17) 20–?–40; (18) 54–?–21; (19) 25–?–35; (20) 81–?–24; (21) 64–?–54; (22) 68–?–34.

All of the above figures and especially in the second generation cases *seem* to point clearly to the validity of the phenomenon of antedating or anticipation. A closer examination, however, shows *one great fallacy underlying the statistics and that is, as usual, the method of their collection.* In a great majority of cases *the period of time during which the cases have been collected is not clear.* (for example, in Mott's statistics where no mention is made of the period during which his figures have been collected), but it is generally within thirty years. As the average ancesor is at least that much older than his average descendant it would be practically impossible for the descendant to be older than the ancestor at time of commitment, and the age difference between ancestor and descendant would generally be from 30 years to zero, in favor of the ancestor, and in certain cases where the descendant enters the hospital before the ancestor it would be 30 years or more. This means that in the cases of some of the investigators it has been possible for the younger descendants to enter the hospital, *but time enough has not elapsed for the older descendants to arrive.* As the Taunton figures have been collected since 1854, this objection does not obtain with such force. Nevertheless, *the rate of commitment has increased* within the last generation so that even in my cases the *bulk of the families* has arrived within the last thirty years and thus the factor stated above still plays its part in falsifying the manifest results. Rosanoff (160) puts his objections in somewhat different form as follows.

> Observations like those of Morel and Mott may be accounted for to some extent by the manner of selection of the material. Where neuropathic manifestations are found in both parent and offspring the case is usually one of a person who has led a sufficiently long and successful extramural existence to enable him to marry and to have at least one child—if proper allowance be made for infant mortality one should say, perhaps, at least two children—in comparison with another person not necessarily thus qualified. Now, it is clear if one selected from any series of asylum cases a group consisting of patients who are married and have had at least two children, one would find that the average age of onset of the mental trouble for such a group would be higher than the general average for the entire series; this difference would be in no way dependent upon any relationship such as that between parent and offspring.
>
> To return to Mott's figures, the fact that he found the age of onset to be under 25 years in only 1.8 per cent of the fathers and 4 per cent of the mothers, or that he found the age to be 60 years or over in only 1.7 per cent of the offspring, is hardly to be attributed to any "law of progressive degeneration," or "law of anticipation."

Further, one may ask how many of those offspring who were living and well and still young at the time of the compilation of the statistics may become insane at advanced ages? This question becomes even more pertinent when due account is taken of the almost uninterrupted rise in the incidence of insanity in correlation with increasing age, as shown in Chart XIII.

Still it is hardly to be disputed that, after all probable error in the statistics has been duly discounted, the fact remains that in the majority of cases in which a difference is observed the gravity of the neuropathic manifestations is less and the age of their onset is higher in parents than in their offspring.

A good many opinions have been expressed on this question of anticipation, a large number having in mind the Morel doctrine of polymorphism. Those who favor this doctrine have on the whole been inclined to find anticipation, and in addition an increase in the severity of the disease from generation to generation. Thus Krause (113) believes it to exist, as do Berze (12, 13) and Damköhler. Schuppius (174) and Luther (125) do not find it a prominent phenomenon, nor do the figures of Shlub (173), Jolly (100) or Kreichgauer (114) give it much importance. Rudin finds that this phenomenon is prominent in the brothers and sisters as well as in the children of dementia praecox patients. Rudin (164, 165, 166) makes the point, which is well taken, that it is the beginning of the psychosis that should be counted and not the incarceration into a hospital. This, of course, is the great defect in my figures and in those of Mott.

It, therefore, is probable that when all is said and done, in a very large proportion of cases the descendants of the insane who themselves become insane do so at an earlier age than their ancestors. Moreover, as is well known, these descendants have a much lessened chance for marriage and so this factor of anticipation seems to be, as Mott believes, a potent factor for race regeneration through elimination. One need not, or course, subscribe to any such antropomorphic absurdity as that nature has any intentions in the matter. It is perhaps better to speak of this phenomenon as the downward trend of psychiatric families just as the sane descendants represent their upward trend.

THE POSSIBLE SIGNIFICANCE OF ANTICIPATION AND ANTEDATING

It needs to be pointed out that the main hereditary characters of life occur at nearly the same periods in each generation. We would think it highly remarkable if the time of walking was greatly

irregular and in fact the average parent commences to worry and with reason if the child delays six months in the time of acquiring this great character. Similarly with talking. The school periods are on the whole adjusted, though crudely, to a uniformity in development, and in fact the child that is either retarded a couple of years or advanced a similar period is looked on as exceptional. The eye color develops at the same age, the menses are established at from 12 to 15 years in the overwhelming majority of cases, and the cessation of the menses occurs within a relatively few years for all but a very few women. The greatest height in men is reached around the early twenties, though in some races there is a somewhat earlier full development than in others, but even then the difference is but a year or two.

There is, however, wide variability in other characters. The sex powers in men vary enormously, in that some establish virility at an early period and lose it late, thus having a very wide span of sexual activity, while others establish virility late and lose it early, the difference being as much as a quarter of a century, while the difference in actual power may be even more conspicuous, ranging from those whose power is exhausted with a single act with the lapse of days to the next possible repetition to those whose power and desire are almost inexhaustible and offer the occasion for much discussion in smoking room and boudoir. The variability in the time of declining of eyesight, of memory, the onset of tooth decay, of baldness is remarkable, but the qualities discussed are specific, non-generalized qualities. That is, some men lose hair early but retain teeth and eyesight late, nor is the retention of sex power at all related to mental virility or to retention of memory. It is true there is sometimes a correlation in the decline or retention of these powers and qualities but generally there is not. For example, the feeble-minded of low grade usually have poor teeth but retain their hair exceedingly well.

When we speak of early loss of teeth, hair, eyesight, and sex vigor we are discussing situations more resembling *disease* than true hereditary qualities, or rather we discuss *individual* peculiarity more frequently than *family failing; we are considering phenomena that can be duplicated by disease conditions or by environmental factors.* Thus early decay of the teeth follows rickets, syphilis, bad diet, etc., and early loss of hair has similar antecedents; early loss of eyesight

follows poor lighting conditions, general loss of vigor, sickness, etc. Sexual impotence at an early age follows disease of the cord, arteriosclerosis, over-indulgence and over-stimulation, etc.

There is therefore some ground to suspect that environmental conditions, social status and disease play a rôle in bringing about such variable conditions and that while the matter may be constitutional[2] it is not heredity but individuality which must be considered.

The marked variability in the age of onset of these mental diseases, the phenomena of anticipation and antedating thus point also to the conclusion that we are dealing with disease rather than the kind of heredity involved in the main biological characters.

[2] In again pointing out constitution and heredity are not at all necessarily related I am emphasizing a point well made by the German writers, Bauer (9), Baur (8), Fischer (8), etc.

CHAPTER IX

The Transmission of Specific Mental Diseases

The term "vertical transmission" is here used to imply the transmission from one generation to another, just as horizontal transmission will be used to mean the appearance of mental diseases in the members of the same generation. I have given myself these simple clinical problems.

1. Vertical transmission of mental diseases. If an ancestor, a parent, have a mental disease of a certain type what type of mental disease is likely to appear in his "insane" descendant? This does not state that he is bound to have insane descendants—I know indeed that he is not bound to—nor does it make any inferences as to the number of insane descendants he will have, and it leaves out of account the most important research, what becomes of the non-asylum descendants, some of whom will have mentally sick descendants and some of whom found "normal" lines. Nor does it state at all positively that *because* the ancestor has a certain mental disease does the descendant have his type of disease. On the contrary no causal relationship can be thus established, for it is more likely that some antecedent situation operates to produce the particular disease found in parent and child.

2. Horizontal transmission of mental diseases. If a brother or a sister have a specific type of mental disease what type of mental disease will another brother or sister be likely to have, on the basis of asylum cases? Again I must limit my results, they do not state anything of the non-asylum brothers or sister's many of whom so far as I can see on the basis of private practice and experience elsewhere, are to all intents and purposes normal. (Here I must be emphatic: once we know that mental disease has occurred in a family we scrutinize the other members of that family with a prepossessed and prejudiced eye, we tend to magnify into psychopathia that which otherwise we would call mere individuality.) And surely the mental diseases of brother and sister are not necessarily evidences of pure hereditary cause, for they may arise from the same germ plasm injury

in the antecedent generation or from similar environmental causes in the way that syphilis and tuberculosis affect members of the same family.

These are the simple clinical problems to be discussed in the following pages of part II. Based on records of varying degrees of perfection and on hospital examinations at a time when psychiatry has not established positive diagnostic criteria, I can claim no finality for the conclusions reached. But at least clinical problems have been attacked in a clinical manner, surmise and prepossessing theory have not interfered too much, and the conclusions reached pretend to no solution of the deeper problem of heredity.

To answer these questions I here adduce all the case wherein three or more generations are represented, a part of the cases in which two generations are represented, and a part of the cases where 2 or more members of one generation are represented. In the three generation cases direct heredity is not always present, and in the majority of cases only the first and third generations have been represented in the hospital, but in the most of these cases we have been able to obtain fairly satisfactory accounts of the missing links in the hereditary chain so that the data have a distinct, though not by any means ideal value. The two generation cases have been selected from the great mass of the total number of cases, not because of any peculiarity in the cases themselves, but because either the records in these cases are better or else some member of the family is at the present time in the hospital so that investigation has been more satisfactory. In other words, there has been no selection of cases to prove or disprove any point.

Key to charts

Square.....Male
Circle.....Female
I (in square or circle).....Insane—not in Taunton State Hospital
Number (under square or circle). Patient in Taunton State Hospital
D.P.....Dementia praecox
M.D.....Manic depressive insanity
S.D.....Senile dementia
Paran.....Paranoid
Involl.....Involution
N (within square or circle).....Normal
Al.....Alcohol
T.B.....Tuberculosis
+.....Died

(For their work in reproducing my sketches of the family trees, I am under deep obligation to Dr. Chas. S. Lipschutz, then a student at Tufts Medical School, and to Miss Marian Sweet my former laboratory assistant.)

It is remarkable that with all the discussion on the heredity of insanity no other investigator to my knowledge has been able to adduce a family with four generations of hospital cases. Other hospitals are older than Tauton, other communities are as settled. The rarity of such authentic, many generation cases is significant. *Not many cases of 4 generation mental disease can be adduced because it is rare for mental disease to last 4 generations in any one group.*

FAMILY GROUP NO. 1, REPRESENTING FOUR GENERATIONS

A. *First generation.* Hospital No. 247. Female, married, 46 years old on entrance May 26, 1854. The record here is scanty. Six months insane, first attack. She lost her appetite, grew thin; "not violent, suicidal or talkative; not hereditary; very melancholy."

February 1, 1855, "no change, very melancholy. Sits around."

May 10, 1857, discharged, "very much improved." Died later of cancer; is said to have been more or less depressed the rest of her life.

Husband said to have been normal, though he drank a little.

B. *Second generation.* Hospital No. 4657. Son of the foregoing; single, laborer, 28 years old on entrance May 15, 1872. His father died of pneumonia just previous to this attack. Always religious and reserved. Ordinary mental capacity; had poor education; occasional depressed spells since the age of 20. Onset said to have followed worry over father's illness. Sleepless; excited; preached and prayed; refused to eat; became apprehensive; feared harm. Was tube fed for some time, then became actively excited for several months; destructive to clothing. Later on became clownish; said he was Jesus Christ, in facetious manner.

In November, 1873, became quieter and from then on improved, and on June 27, 1874, he was discharged, "recovered."

Second admission. Fifty years old on entrance February 8, 1892. Since his discharge in 1874 said to be well; worked at his trade of machinist, earning good pay; was sober and steady. Became depressed about his spiritual welfare and came to the hospital voluntarily for advice. Coherent; memory perfect; orientation perfect; no definite hallucinations; no distinct delusions; disinclined to eat; depressed. Recovered gradually and was discharged April 15, 1892. Diagnosis, acute melancholia.

Third admission. Fifty-five years old on entrance February 11, 1898. Told physicians, "Jesus Christ is in Taunton and is going to Billy Martin's for a cocktail." Accused people of poisoning him. Showed violence to police officer who had attempted to arrest him the night before.

On entrance, exhilarated, profane, good-natured. Dances and attempts to sing. Says he would like to be a clown. Talks to imaginary persons.

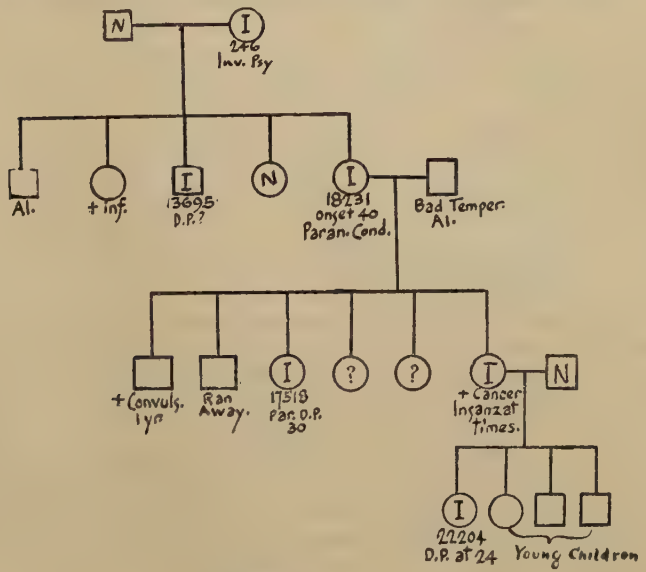
Accuses himself of all sorts of vile relations with animals. An incoherent and loosely associated flow of thought.

Several months later, often seen sitting at window and talking to imaginary persons, but denies hallucinations. Has become reserved, quiet, and tractable.

July 5, 1898, very tractable, volunteers no conversation, mutters to himself. Seems indifferent.

February 10, 1899, exalted delusions, has a mint of money "as old as the earth;" "will not tolerate questions but retorts with a string of mysterious, scientific, and unanswerable questions, then turns away as if he had silenced the questioner, or overawed him by his learning."

In 1899, works in the brush shop, gradually becoming demented.



FAMILY NO. 1

In 1900, surly, indifferent, asks meaningless questions with a haughty air.

In 1904, mute, never takes initiative in conversation, monosyllabic answers; appetite and sleep are good.

In 1908, had a short, excited attack with flight of ideas and talkativeness.

In 1911, signs of old age were prominent; was disoriented; poor memory, demented, and died December 21, 1911, of erysipelas. The hospital diagnosis at the end of his life was "dementia praecox."

C. *Representing second generation.* No. 7247. Daughter of A, sister of B; married; 40 years old on entrance July 23, 1877. Sudden onset following illness. At times violent. On entrance quiet, very frail appearing decidedly sick. She gradually improved physically and likewise mentally. Discharged

September 21, 1877, recovered. During stay in hospital was mostly depressed, no other symptoms recorded.

Second admission. No. 18231, 70 years old on entrance April 17, 1908. Had been in the almshouse for 10 years before admission. Had insomnia and excited periods during that time. At intervals confused; had the habit of stripping herself of her clothing. In hospital, careless, untidy, excited; memory good. Very circumstantial in replies. Has decided delusions of persecution directed against the almshouse people; fantastic sexual delusions.

Later, in 1908, delusions against the Catholics (she is a Catholic). Very suspicious and actively hallucinated; noisy.

In 1909 excited at times, hostile, irritable. Discharged to State Board of Insanity for family care. Died in 1915, condition unchanged.

D. *Representing third generation.* No. 13558. Daughter of C; married; 33 years old on entrance September 1, 1897. Onset at 31; has been a heavy drinker at times. Married early. Limited mentally. Convulsions in childhood.

In hospital, worried, depressed, neighbors have persecuted her by the way they arrange their blinds, thus alluding to fact that she had lived with her husband before marriage. The priest is conspiring with her husband for a divorce. (The attitude of the Catholic Church towards divorce, of course, makes this ridiculous.) Later, excited, restless, attempts to escape. Has hallucinations of hearing; suspicious.

April 6, 1898, discharged to husband, somewhat improved.

Second admission. No. 17588. Forty-one years old on entrance December 21, 1906. Had been in the Butler Hospital (private hospital) for six months and in the Rhode Island State Hospital six months. It is reported by these hospitals, "orientation good, indifferent, retarded, depressed, poor attention, fair memory. Persecutory ideas prominent."

In this hospital depressed and yet apathetic; delusions of persecution; suspicious. Discharged against advice June 12, 1907, "not improved."

Third admission. No. 20404. Forty-nine years old on entrance October 3, 1912. No change. Delusions of persecution; eccentric conduct; memory now somewhat defective. Discharged against advice. Diagnosis at this time, dementia paranoid.

Has been at home since, does not work, hostile, jealous, untidy, deluded, and destructive. Occasionblly is very excitable.

This patient had a sister who was at times insane, and always was peculiar.

E. *Representing fourth generation.* No. 22204. Great granddaughter of A, niece of D; married; 24 years old on entrance October 21, 1915. At present in hospital. Father is alive, said to be normal. The mother died of cancer, at times insane. She has two brothers and one sister, all younger than she, all said to be well. Had ordinary mental capacity. Married at the age of 23. The onset of the present illness three weeks after the birth of the first baby; was very excited; filthy. At times very humorous and facetious. Runs around the ward, shrieking, screaming, and tearing the clothing of other

patients. Reacts distinctly to auditory hallucinations. At times it was necessary to tube feed her because of a fixed belief that the food was poisoned. After six months the excited period passed off; became very quiet; very silly; grimaces; answers in foolish and unintelligible manner. Replies irrelevant. Does no work. No hallucinations or delusions can be elicited. Extremely apathetic, "bench type." Diagnosis of hospital, at present time, dementia praecox. There was some question as to whether or not the first episode was a catatonic episode occurring in manic-depressive. At the present time the patient acts distinctly like an apathetic dementia praecox.

Summary of this family: First generation. A, very difficult to diagnose, but probably an involution melancholia type.

Second generation. B had three admissions, in the first two of which he acted very much like a maniac, the second admission particularly, apparently a simple depression. In the third attack he started with extremely expansive ideas, passed quickly into apathy, mutism, paranoid ideas, and dementia. C, a sister, second generation, had an acute psychosis following sickness at the age of 40. From 60 on showed paranoid ideas, periods of confusion, excitement, and disorder—character change prominent. No dementia. In this generation also there were one alcoholic brother, one sister who died in infancy, and one sister, the youngest, who seems to have been the only sound person. She, however, is said to have been over-pious.

In the third generation there were six descendants. One, patient D, had a paranoid psychosis starting at the age of 30 and apparently of the dementia praecox type. There was one brother who died of convulsions, one sister died of tuberculosis, one brother ran away, one sister insane at times, one sister, the mother of the patient of the next generation was insane at times and died of cancer.

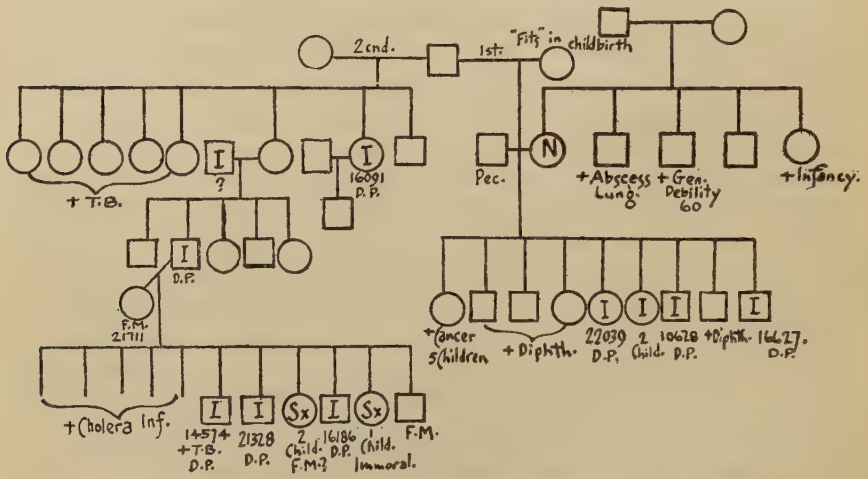
In the *fourth generation* four descendants, all young people. The oldest developed a psychosis at 24, after childbirth. Catatonic symptoms at first with a trend that resembled manic-depressive insanity (facetiousness). Final picture that of dementia praecox.

While there seems to have been a gradual worsening in the type of the psychosis and the irregular appearance of anticipation in this family, there does not seem to have been any such transformation of psychotic type as that depicted by Morel. The psychoses appear to have passed from depressions and excited periods over towards dementia praecox (paranoid), and finally dementia praecox, catatonic type. In other words, while there has been change from generation to generation, the change, as will be shown later, has followed in a definite direction.

The family groups to be now detailed belong to what I call the "three generation families," in that individuals of three generations are represented in the hospital records. Few cases of three hospital generations are cited by other writers.

FAMILY GROUP NO. 2

This is unique, in that one person, a female, is the ancestress of two separate groups of people who found their way to this hospital. In the first instance, she is the maternal aunt and grandaunt; in the second, she is the aunt. The two groups will be presented successively.



FAMILY NO. 2

A. *Representing first generation.* No. 10678. Female, married, 51 years old on entrance November 12, 1889. Pedigree (see chart). Daughter of a couple who had six other daughters and one son. Five of these daughters died in early womanhood of tuberculosis, leaving behind them families. The one other daughter who did not die early is the direct ancestress of the other patients of this group. The brother was an alcoholic. The father of this patient by a previous marriage had one son. This half-brother is the direct ancestor of the insane family group to be detailed later.

Onset at 47, at menopause. Had attacks of sleeplessness and excitement. On entrance, delusions of persecution; violent; hallucinations of hearing, sight and smell. Delusions of reference along sex lines prominent. "Room had been filled with horrible odors." Delusions of poisoning. Suspicious; apprehensive; coherent conversation; memory practically intact. Improved rapidly; discharged February, 1890, for trial.

Subsequent history can be briefly summarized. She had three further commitments; No. 13350, March to June, 1897; No. 13599, June 1897, to June, 1899; No. 14239, July, 1899, to January 6, 1904, when she was sent to Medfield State Hospital. Each attack was characterized by excitement, paranoid delusions, and hallucinations. The attack would last about two or three weeks and then would replaced by a quiet, apparently normal period. During each stay in the hospital this alternation of paranoid attack with normal state was repeated two or three times. Finally, these short attacks became so frequent that she was transferred to Medfield, where she died, condition unchanged. Never depressed; no distinct dementia even when she became old. The hospital diagnosis was manic-depressive insanity. This seems to me to be an error, as the attacks were distinctly paranoid and attended by exceeding definite hallucinations. No real flight of ideas is recorded. A circular type of dementia praecox seems more in accord with the facts.

Between this patient and the other members of this family who came to this hospital is a nephew, who at 35 committed a murderous assault, was adjudged insane, and died in the Bridgewater State Hospital for Criminal Insane. Diagnosis, dementia praecox.

B. His wife, the mother of the succeeding patients, No. 21711, was admitted to this hospital November 7, 1914, at the age of 56. She had tuberculosis of the lungs and presented no signs of "insanity," but was thought to be somewhat below par mentally though not definitely feeble-minded. She died May 12, 1915, of tuberculosis.

C. *Representing third generation.* Their son, grandnephew of A, No. 14574, single, 21 years old on entrance May 5, 1900. Decided and plain case of dementia praecox, incoherent, delusions of persecution, hallucinations of sight and hearing, apathy, profound dementia. At 27 died of pulmonary tuberculosis February 5, 1906.

D. No. 16186. Male, brother of foregoing, single, 25 years old on entrance November 12, 1903. Mental status same as brother's. Discharged to Medfield State Hospital March 26, 1909, still there. Dementia praecox.

E. No. 21378. Female, sister of foregoing, 34 years old on entrance May 23, 1914. Condition about as brother's, except that dementia is less rapid and paranoid ideas more marked.

One brother is in the Waverly State School for Feeble-Minded. Two sisters are feeble-minded and immoral. Five brothers and sisters died in very early infancy of cholera infantum so that *of this third generation every member who did not die in early infancy became "insane" or else feeble-minded.*

FAMILY GROUP NO. 2, SECOND BRANCH

A. *Representing first generation.* We start here with the same woman A, of the foregoing, No. 10678, whom I have diagnosed as circular dementia praecox.

Her half-brother, same father, and whose mother is said to have had eclampsia, died at the age of 60. He is said to have been peculiar all his life and insane towards the end. His wife came of a normal stock and died at 74 of cancer of the uterus.

B. *Representing second generation.* No. 16627. A nephew of A, and son of her half-brother; single, 24 years old on entrance April 17, 1911. Typical dementia praecox of paranoid type, self-center, peevish, very marked delusions of persecution and reference, rapid dementia and apathy, somatic delusions, masturbation. Transferred to Foxboro.

C. Brother of the foregoing, No. 10628, 14 years old on entrance August 19, 1889. Masturbation, morbid sexual feeling, strong religious feeling, self-accusatory and depressed, became mute and apathetic, occasional periods of excitement, occasional mannerisms. Became demented, died July 21, 1893.

D. Sister of foregoing. No. 22039, 54 years old on entrance June 23, 1915. In hospital at present. Restless and noisy at times; delusions of persecution and poisoning. Hears voices, fantastic sounds. Hears people being tortured and roasted. Sees children thrown out of window. Went to police for protection against enemies. At present is apathetic; memory good, markedly deluded, and hallucinated, occasionally excited, very indolent; no distinct dementia.

In addition to the members who have appeared in the hospital there were four brothers and sisters who died in early infancy of diphtheria. Another died of cancer, one sister is in the Westboro State Hospital, and is the only one who married. Has two children; was insane at 40. Diagnosis, dementia praecox. There seems to have been but one type of psychosis present in all members of this family. The ancestress had a psychosis that more nearly resembled circular dementia praecox with prominent paranoid ideas, and all her descendants in the two directions suffered from dementia praecox, with a strong paranoid trend.

The chart of this family might easily be labeled, "Why Insanity Is on the Increase." It will be seen that in the first generation the large majority died of tuberculosis. In the third generation all who did not die of infantile diseases became insane. The tendency of all modern hygiene is to eliminate tuberculosis, and in so far as possible to lower the death rate in infancy and early childhood. Such a process as has often been pointed out can only mean that certain abnormal individuals are saved from early death only to developmental disease in later life.

The above quoted paragraph is as written in the original publication—1916. Today I believe that while there is point to the observation it neglects the following—that while the potential cases of dementia praecox and feeblemindedness are saved by modern hygiene, the potentially fine, normal, and desirable are also saved, that modern hygiene thus saves the assets of the race as well as its liabilities. I have known very fine children of fine families to die of tuberculosis.

Further, there is the question of germplasm injury by tuberculosis, and other infections, not lightly to be dismissed and which gives to modern hygiene a great eugenic value. Of this, more later.

FAMILY GROUP NO. 3

A. *Representing first generation.* No. 13252. Female, a widow, 69 years old on entrance December 26, 1896. Onset within six months. Suicidal, tried to burn her house down; hallucinations of sight and hearing; noisy and restless at night. Believes her father and brothers, who are dead, are after her. Is suspicious, irritable, and resistive. Became demented and died of lobar pneumonia April 4, 1904, aged 77. Diagnosis, senile dementia.

B. *Representing second generation.* No. 12072. Is a niece of A, sister's child, age 52 on entrance, January 22, 1894. Mother died of fright after a fire (probably insane). Father died of sunstroke. One brother had "brain fever." Other fraternity negative.

Onset at 42. Has become disagreeable, forgetful, excitable, depressed. Believes a spell is being cast over her. She is being injured. Her relatives fill her closet with filth and feces and destroy her things. She hears voices calling to her. In June, 1894, discharged improved. Paranoid condition?

C. *Representing third generation.* No. 19419. Female, a daughter of B, a greatniece of A, single, 38 years old on entrance October 20, 1910. This is the third attack. First at 23, lasted six months; the second at 32, lasted six months. In each of these she was nervous, could not work, sat about all day, felt she was not understood, was supicious.

In this attack is suspicious; people are watching her and are talking about her; self-accusatory; threatens suicide; negativistic. At times violent, surly and resentful. No hallucinations admitted; no distinct dementia. During stay in hospital health gradually failed. She was found to have pernicious anemia. She was sent home to die December 10, 1913. Paranoid condition.

Summary. In the three generations a rather similar condition is found in all the patients with a distinctly earlier onset in each. In the first, the senium ushers in a paranoid psychosis; in the second, a similar disease occurs at the involution period; while in the third, the psychosis starts in early adult life.

FAMILY GROUP NO. 4

A. *Representing first generation.* No. 1412. Male, widowed, 70 years old on entrance January 18, 1860. Stock unknown. Has been insane for four or five years; troublesome, cross, quarrelsome. Hauty and suspicious in attitude. No particular delusions; not suicidal; at odds with everyone. Died May 29, 1867.

Wife a normal woman. There resulted from their union two males and five females. One male was insane in old age, one is B in this group. Of the

females, one died at 85, insane. One was the mother of C, and insane, while the fate of the rest is unknown.

B. *Representing second generation.* No. 15759. Son of the foregoing, 71 years old on entrance December 24, 1902. Considered of average intelligence, seclusive and peculiar, a poor worker. At 71, the first attack. Was nervous, suspicious, excited, and unreasonable.

In the hospital had a markedly disconnected conversation. Very noisy; decided flight of ideas; distractible; keen and witty; elated and sleepless; destructive and mischievous. Apparently a typical manic.

Remained insane until death two years later, November 21, 1904.

C. *Representing third generation.* No. 17986. Niece of foregoing sister's child, a granddaughter of A. Single, 62 years old on entrance October 20, 1907. At present in hospital. Father was a peculiar man. The mother was considered bright. She did fairly well in school but did poorly at work, invariably discharged. Never capable of earning a living or caring for herself; lazy and untidy. Relatives supported her. She had periods of active mental disturbances for many years.

In the hospital, hallucinations of hearing, talks to imaginary persons, poor judgment. Very marked but incoherent delusions of persecution against people in the environment. Marked sexual delusions, very troublesome, stubborn, and irritable.

In 1916, condition worse. Hospital diagnosis, paranoid dementia praecox.

Summary. In the first generation paranoid delusions, developing late in life. Second generation, at least three insane, one with a very decided manic attack at 71, one senile psychosis, and one with unknown psychosis. Third generation, a paranoid dementia praecox of very slow course associated with moron intelligence. Anticipation not prominent. Remarkably late hospital commitment in three generations.

FAMILY GROUP NO. 5

In this group are three people—in the first generation, a female, in the second generation her son-in-law who is of course not reckoned as of same stock, and in the third generation his daughter and her granddaughter.

A. *Representing first generation.* No. 8614. Married, 53 years old on entrance August 3, 1882. Comes of a family notorious for immorality, poor intelligence, and intemperance. Psychosis of rather rapid onset, of one year's duration.

In hospital believes her body is united with those of persons long dead. Her household is cursed by God. Hears spirits of dead and absent friends. Eats and sleeps poorly; apprehensive; inclined not to move or talk though occasionally violent and noisy; gradually demented.

In 1884, it is stated she is apathetic, believes herself surrounded by fairies who operate on her. She is in constant danger of her life.

February 21, 1895, transferred to Tewksbury.

Her daughter, who is said to have been somewhat less than the average in intelligence went through life without incident. She married:

B. *Representing another first generation.* No. 6511. Twenty-one years old on entrance April 16, 1866. His father and mother lived to old age. The rest of the family are said to have been ne'er-do-wells. Onset, 18. Occasionally excited, noisy and apprehensive. Became actively excited, destructive, and filthy. Had to be tube fed, recovered slowly. Discharged July 14, 1877.

Second admission. At 29 years, November 23, 1883. Excited, noisy, incoherent, profane, destructive to clothing, silly in manner and speech. Remained silly, seclusive, and incoherent until July 2, 1885, when he became decidedly catatonic, mute, tube fed, and *lay with eyes closed for seven years* when he commenced to improve and was discharged December 3, 1892, improved. He died in 1897. From his marriage with the daughter of A, there resulted:

C. Who thus represents a third generation on A's side and a second generation on B's side. Female, single, 17 years old on entrance April 15, 1902. Always feeble-minded. At 15 raped by strange man. Began to grow nervous, seclusive, irritable. Imagines she is pregnant, though menstrual periods are O.K. Evasive, mute.

Later note says, dull, apathetic, rapidly demented, and died of tuberculosis January 15, 1907.

Summary. With two insane stocks it is difficult to analyze the situation. B and C suffered from very much the same type of mental disease though the psychosis is more marked in the daughter's case and attended by feeblemindedness. If we regard A and C we are dealing in the case of the grandmother with a paranoid psychosis of middle life which may be called presenile dementia or dementia praecox. The granddaughter had hebephrenic dementia praecox at a very early age. There is, therefore, evident a worsening of the disease, but no new character except a possible feeblemindedness has appeared, and essentially the psychosis has not altered. The feeblemindedness very likely is that early dementia praecox discussed by Kraepelin.

FAMILY GROUP NO. 6

In this group the descent in each case is indirect; that is, through a niece. However, we have a good history of the other members of the family.

A. *Representing first generation.* No. 13096. Male, married, 66 years old on entrance August 11, 1896. Family history is negative. He was a soldier in the Civil War and Indian Wars. He had two paralytic shocks at 66; since then has been excited and depressed. Talks of fighting the Indians. Markedly hallucinated; memory poor; very violent; and at times homicidal. Occasionally expresses grandiose ideas. Talks of running for public office. Has albumen in urine. Clinical diagnosis, chronic interstitial nephritis. Wanders about the ward confused. Was discharged to the Medfield State Hospital March 16, 1897, where he died shortly afterwards. Diagnosis, "arteriosclerotic dementia."

He had three brothers and four sisters whose history is not known. One of these brothers, himself said to be normal, was the father of:

B. *Representing second generation,* in the direct line. No. 11802. Female, married, 27 years old on entrance April 28, 1893. Illness of child caused the onset. Was incoherent; attention poor; memory good; depressed. "All her troubles came upon her because she did not take a physic. Oh, that such trouble should come because one did not take a physic." Motor excitement; reiterates automatically, clapping her hands: "My God! How could I do it, only 28 years old." Is in constant motion, walks to and fro in the ward. Destroys her clothing and the bedding.

In July, 1893, improving, and end of July, 1893, discharged, recovered.

Second admission. No. 14979. Entered May 7, 1901. Illness of child is cause of onset. The child has tuberculosis. "My blood has turned to water" Memory good; orientation exact; no flight of ideas or retardation; no hallucinations. Later became markedly depressed and agitated; said "she exudes poison through her skin and is the cause of sickness and death in the community." Became destructive and suicidal. Discharged against advice October 1, 1901, improved.

Third admission. No. 19082. Forty-four years old on entrance February 9, 1910. Since then in hospital. Got along well at home. Operated for torn cervix and then became talkative and restless.

In hospital, restless, agitated, exactly same delusions as before. Intellect clear.

In 1910 and 1911, filthy, soils clothing, very foul and profane.

In 1916, memory intact; orientation good; no hallucinations. Delusions, as before, of somatic nature. Very immodest; chronic masturbator; very untidy. Emotionally, occasionally agitated, but usually indifferent. Does not work.

Diagnosis: The first two attacks are exactly like an agitated depression. The last attack has lasted six years. There is present apathy, with some signs of deterioration. She married twice. One child died of tuberculosis. There were two miscarriages and one young child is apparently normal.

She was one of seven sisters. Five have large families. One died in childhood; one died of cancer; one died in childbirth. The oldest sister, a confirmed alcoholic, is the mother of C, and her husband deserted her because of her difficult character.

C. Representing third generation. No. 20511. Niece of B. Married, 35 years old on entrance January 8, 1913, and is in the hospital. Operated on for cancer of the breast. Since then, delusions of reference, persecution, hypnotism. Hallucinations of sight and hearing; vague ideas of grandeur; seclusive; fantastic delusions. Decidedly a paranoid dementia praecox.

Summary of family. In the first generation, psychosis following paralytic shocks. Second generation, a brother's daughter, three attacks—first two, agitated depression, the third difficult to diagnose and having features of manic-depressive insanity and of dementia praecox. The third, paranoid dementia praecox. In all three cases, somatic disease or external incidents play a part. No death of stock is noticeable. It is possible that coincidence rather than heredity is the source of this family's mental disease.

FAMILY GROUP NO. 7

A. *Representing first generation.* No. 1235. Female, 55 years old on admission May 11, 1859. Mother died "insane," father alcoholic. Insane since 48. Demented the last two years. For the last four weeks noisy, violent, and excited.

In hospital, generally demented; quiet, filthy; destructive of her own clothing; incoherent. Died October 31, 1875. Apparently dementia praecox.

B. *Representing first generation.* No. 10536. A sister of the above, 60 years old on entrance March 30, 1889. Onset at 60. Is depressed, apprehensive, sleepless; believes that the persons around her are going to kill her; agitated. Died April 9, 1899. "Involution Psychosis."

C. *Representing second generation.* No. 17508. Daughter of A, niece of B. 68 years old on entrance October 15, 1906. Father is alcoholic. The patient was born in the almshouse. Married at 18, has three children; said to have been bright and capable. For many years she has had attacks of excitement and periods of melancholy. Later, the excited periods are longer, at such times talkative, obstinate, abusive, and violent. Last four years has been circulating scandalous stories.

In the hospital, is oriented; memory intact; very vile and abusive. Delusions of persecution against many people, not systematized. Marked sexual ideas directed against others. At times has hallucinations of hearing. Believes babies are killed in the hospital. Grew senile and demented. Died May 26, 1913. Paranoid condition, possibly paranoid dementia praecox.

D. *Representing third generation.* No. 13763. Female, married, daughter of C, 30 years old on entrance April 8, 1898. Death of youngest child precipitated the attack. Became depressed, mute, prayed a great deal, no appetite. Threatened suicide.

In the hospital, a drawn and fixed expression. No other catatonic symptoms.

April 16, 1898, brighter. Hears voices talking to her. Refuses to tell what they say. Commencing to eat.

June 29, 1898, is in the fourth month of pregnancy. Discharged recovered. Died four years later. Probably catatonic dementia praecox, possibly depressed manic.

Summary. In the first generation in the direct line, probable dementia praecox with excited periods throughout life, a sister having involution psychosis of some type. The second generation, a paranoid condition, strongly resembling paranoid dementia praecox. In the third generation, an episode of catatonic dementia praecox. No well-defined anticipation, the condition running without much change from generation to generation.

FAMILY GROUP NO. 8

A. *Representing first generation.* No. 13207. Female, married, 68 years old on entrance November 19, 1896. The father died in a fit (?). Mother died of paralytic stroke.

Patient has been in present condition, in a lesser degree, for 40 years. Is deaf but hears voices talking to her about dead people and funerals. Believes that the neighbors stoned the house and abused her. Her answers are coherent. Conduct is childish. She has entertained mild paranoid ideas for years. Has occasional flare-ups of excitement. Sent home after one month, probably old, slow-going dementia praecox. Married a moderate alcoholic.

B. *Representing first generation.* No. 10073. A sister-in-law of A (husband's sister). Female, 62 years old on entrance May 12, 1883. Two brothers and one sister insane. Insanity has come on within a year. Indefinite delusions of persecution and approaching injury. Violent, noisy, destructive. Has hallucinations of sight and hearing. Is evasive and irritable. Memory defect marked. Died. Senile psychoses, probably senile dementia.

C. *Representing second generation.* No. 13208. A daughter of A and niece of B. "Insanity," therefore on both sides. Single, 41 years old on entrance November 19, 1896. Is feeble-minded. Has had epileptic attacks for years. Vicarious menstruation through nose. Same delusional ideas as mother but more marked. Conduct disorder greater.

Readmitted, No. 15200, 46 years old on entrance October 21, 1901. Very violent and abusive. Delusions of reference. Hallucinations of hearing; insomnia. Sexual ideas and poisoning ideas marked; incoherent; gradually failed. Died January 15, 1902. Dementia praecox.

C. had a sister who was also insane but not in the hospital. This sister had one daughter.

D. *Representing third generation.* No. 12100. Married at the present time, in hospital, 31 years old on entrance March 31, 1894. The father was a moderate alcoholic, mother insane. At 30, ideas of reference and persecution of a non-systematized character. Hallucinations of hearing; generally depressed; occasionally suicidal and excited.

In hospital, became somewhat more cheerful. Was discharged December 23, 1894, slightly improved.

Second admission. No. 15296, June 7, 1902. Never well in interim, but quiet. Symptoms became prominent. Practically the same symptoms but more marked. Memory more defective; marked apathy. Became a boarding-out patient. Symptoms again became prominent in 1910. Quiet, had been boarded out again. In 1916, is demented; has marked apathy; delusions and hallucinations are fading out. Dementia praecox, paranoid, present diagnosis.

Summary. First generation, on one side slow-going dementia praecox; on the other, indirectly, senile dementia. Second generation, feeble-mindedness, epilepsy, and paranoid dementia praecox in one individual. Third generation, dementia praecox, paranoid. In these cases the type of disease has changed but little in three generations. There is no evidence of anticipation except perhaps in the second generation where, however, there has been insanity on both sides.

FAMILY GROUP NO. 9

Of this group little is known. The records are very meager, but they are given for the sake of completeness.

A. *Representing first generation.* No. 2065. Female, a widow, 68 years old on entrance October 30, 1862. Became first insane at 54 and was twice at Worcester Insane Hospital. Noisy, excited, and destructive. Discharged March 17, 1863, improved. Had two further admissions, in 1863 and in 1865, when she died of heart disease.

B. *Representing second generation.* No. 2585. Daughter of A, 32 years old on entrance June 30, 1865. Has been insane for years. Discharged improved.

C. *Representing third generation.* No. 3137. A niece of B, granddaughter of A, 17 years old on entrance November 13, 1867. Discharged November 18, 1869, recovered.

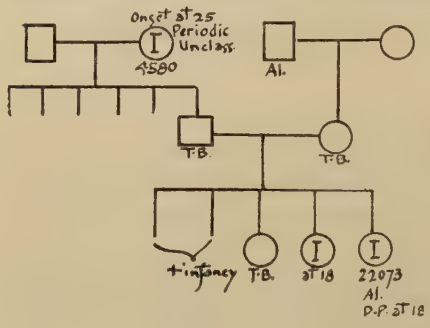
Since the records are so meager no diagnosis is possible. On the face of them anticipation has been prominent, and also the fact that the psychoses in each case belonged to the periodic type rather than to a constant chronic type.

The following 11 groups are of cases representing a first and third generation. The second, though not represented at the hospital, is fairly well known and definite statements concerning it can be made in most cases:

FAMILY GROUP NO. 10

A. *Representing first generation.* No. 4580. Female, married. Entered the hospital March 14, 1872. Has been noisy, excited, and restless with each of four pregnancies, the first in the twenties. Immoral before marriage.

This patient had three subsequent commitments to this hospital. Was finally transferred to Worcester Asylum where she died. From 1872 to 1910 she was out of hospital only four years and never made a complete recovery. The following summarizes her case: Periods of motor excitement, insomnia, destructiveness, confusion, and incoherence follow each other throughout her stay. These were the most prominent symptoms in the first attacks. Later, sexual ideas began to play a rôle, delusions mainly expressed being that the hospital was a house of ill fame and that she was brought there for immoral purposes. At times she refused to eat and in general was hostile to her environment. Hallucinations never prominent. No dementia whatever. She had six children by a normal man. There was much tuberculosis amongst them, but little else is known as they either died early or disappeared. One was the father of the patient B. He married a woman who was a daughter of a markedly alcoholic man. Both he and his wife died of tuberculosis before 30 and he died before his daughter B was born.



FAMILY NO. 10

B. *Representing third generation.* No. 22073. Female, married, 19 years old on entrance July 17, 1915. Of her five brothers and sisters, two died in infancy, one died of tuberculosis, one has been insane at 18, and is now in the Kansas Insane Hospital. Diagnosis on her case dementia praecox.

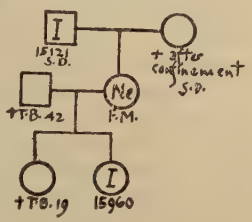
Patient did poorly in school. Broke down at 14; was very hypochondrical. Became depressed, believed her brain was gone. Whines continually about bowels, heart, stomach, and extremities. Dull looking. No hallucinations; memory poor; apathetic. Discharged against advice August 22, 1915. Dementia praecox simplex.

Summary. First generation, an unclassified psychosis with maniacal episodes, later showing delusions of sexual nature. Generally hostile to her environment (paranoid). Second generation, unknown except for marked incidence of tuberculosis and early death. Third generation, early death and tuberculosis, two cases of dementia praecox simplex. Anticipation not worsening of type prominent.

FAMILY GROUP NO. 11

A. *Representing first generation.* No. 1512. Male, widower, 73 years old on entrance August 14, 1901. Onset at 72. Ordinary demented, helpless senile dementia. Memory zero. Died November 6, 1901.

His mate died in confinement at 36 years of age. One of their daughters was the mother of B. She was nervous and unstable, with less than average mental powers, possibly feeble-minded. Her husband, the father of B, died of tuberculosis at 42.



FAMILY NO. 11

B. *Representing third generation.* No. 15960. Female, single, 19 years old on entrance May 22, 1903. Onset at 18, though always feeble-minded. Apathetic, listless, memory impaired, disoriented. Hallucinations of sight, "ghosts haunting her;" untidy; confused; apathetic and indifferent. Marked emotional depression, in time marked dementia. Died September 30, 1906, of tuberculosis. She had a sister who died of tuberculosis at 18.

Summary. In the first generation, senile dementia. Second generation, nervous and feeble-minded person married into tubercular family. In the third generation, tuberculosis and dementia praecox. There seems to be anticipation and worsening.

FAMILY GROUP NO. 12

A. *Representing first generation.* No. 450. Female, married, 26 years old on entrance July 21, 1855. Onset after second confinement. After first confinement had a psychosis for a few days. On entrance, the psychosis had already lasted three months; was noisy and excited. Discharged October 5, 1855, recovered.

Further communications and recent investigations show that she remained well up to recently when she died at the age of 83. Her mate is said to have been normal, but died of consumption at 65.

Their son, the father of B, committed suicide at 23. He was always odd, dissipated, recklessly, contracted venereal disease and while despondent, took his own life. It is positively stated that he was insane.

B. *Representing third generation.* No. 19921. Son of above, grandson of A. Single, 19 years old on entrance December 11, 1911. Was sent in for setting fires. Discharged January 20, 1912.

Readmitted at the age of 23, January 12, 1916. No. 22322. At present in hospital.

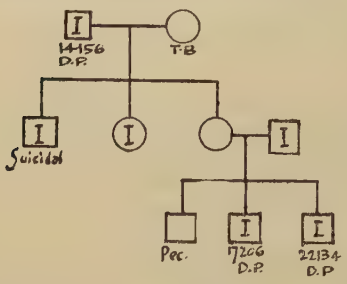
He learned with great difficulty, always considered feeble-minded and "crazy." At 19 set fires. At 23 re-entered as a full-fledged dementia praecox with paranoid ideas. Has well-defined delusions of religious grandeur. He followed a girl in the street, saying that he was fore-ordained to marry her.

Writes letters to the President and to famous and notorious persons of all kinds, offering his services as a statesman, philanthropist, etc. Incoherent. Vague hallucinations of hearing. Diagnosis—dementia praecox with imbecility.

Summary. First generation, puerperal insanity, short attack. Mate tubercular. Second generation, insane, committed suicide, possibly dementia praecox. Third generation, feeble-minded, dementia praecox.

FAMILY GROUP NO. 13

A. *Representing first generation.* No. 14156. Male, widower, 74 years old on entrance May 19, 1899. Had been insane off and on for many years. In two asylums in Switzerland on different occasions. Had a bad temper, was reserved, and seclusive. Became violent and especially so towards daughter. Would allow no one to visit her.



FAMILY NO. 13

In the hospital, depressed, seclusive, excited, negativistic. Talks to himself. As he spoke French only, no good mental examination was made. His attitude was distinctly paranoid and hostile. He died January 20, 1915. Autopsied by writer—pneumonia.

One daughter in insane hospital in Switzerland. One son committed suicide. One daughter, the mother of B and C, said to have been well, but married a man who was insane for short periods. He died at 60.

B. *Representing third generation.* No. 17206. Male, 23 years old on entrance May 17, 1897. Following an unhappy love affair showed failure of memory, excitement, and depression; sleepless; lost weight. Said that some one paralyzed his mind telling him what to do and also what not to do. Has hallucinations of sight and hearing. Threatens suicide. Made a little improvement. Discharged against advice July 3, 1893.

Married between attacks; has three children. The second admission was February 17, 1906. Believes his blood has become poisoned from some bruise or burn. All his trouble originated in that way. He hears men go by who say, "Look at his face," or "It must be changed." People spit in cuspidors and he must clean them out. Has ideas of reference and marked hallucinations. Depressed, irritable. Sent back to Switzerland August 28, 1906. Dementia praecox.

Summary. First generation, a paranoid state, probably dementia praecox. Second generation, very much mental disease. The mother of the third generation married an insane man. Third generation, two brothers, dementia praecox with paranoid trend.

FAMILY GROUP NO. 14

C. No. 22134. A brother of B. Single, 37 years old on entrance August 30, 1915. Condition similar to brother, but is considered more feeble-minded —harmless. Sent on trial visit January 5, 1916. Diagnosis, dementia praecox.

A. *Representing first generation.* No. 269. Male, 70 years old on entrance July 11, 1854. Early life was remarkable. Health feeble. Before entrance had inflammation of the bowels. Became excited four months before entrance for a short period and then recovered. Another attack just on entrance. Very talkative and jolly. Was discharged, recovered, December 4, 1854.

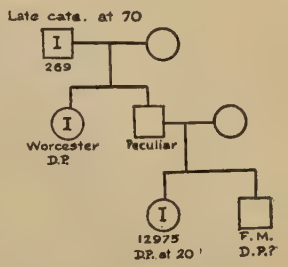
Re-entered March 30, 1855. Sleepless, violent, excited, and destructive. Goes naked and will not eat. Said to have died of exhaustion August 17, 1855. Diagnosis, involution psychosis; first attack resembling manic, second attack either catatonic or depression, perhaps involution melancholia.

One daughter was insane in Worcester State Hospital, then diagnosed dementia praecox. A son, who was peculiar, married a woman said to be of nervous stock and these were parents of:

B. *Representing third generation.* No. 12975. Female, married, 33 years old on entrance March 7, 1896. Had two attacks in girlhood; the first, at 16; the second, at 19. In each she was melancholic, seclusive, depressed with impaired memory. No complete recovery.

She was seduced and married her seducer. At 32, sleepless, lost her appetite, was violent, believed she would be shot; stood mute with eyes closed. Showed excitement and had to be tube fed. Had hallucinations of hearing.

Later note, memory very poor, peculiar in dress and speech.



FAMILY NO. 14

Discharged July 4, 1896, slightly improved.

She had three further admissions to the hospital. Between attacks peculiar, very poor memory, did not work, was jealous and irritable. In each attack excitement, delusions of persecution, hallucinations of sight and hearing, incoherent, and destructive. Sometimes was mute and had to be tube fed. Gradually became worse.

At last report, 1916, she was demented, unable to work, peculiar in conduct. Statements of neighbors that she is worse than she ever has been. Diagnosis, dementia praecox.

Has a brother who is "half-witted," peculiar, and known to be insane.

Summary. First generation, at 70 short attacks, difficult to classify, but having manic and catatonic traits. In the second generation there was one daughter insane. One son peculiar who married a "nervous" woman. In the third generation, as descendants of these last two, were one dementia praecox and one feeble-minded

dementia praecox. There is a gradual worsening of disease but on the whole no marked change in type.

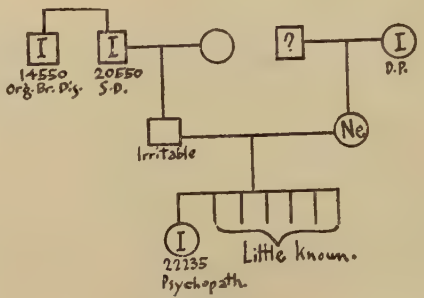
FAMILY GROUP NO. 15

A. *Representing first generation.* No. 14552. Male, married, 69 years old on entrance April 19, 1900. Onset at 68, after hemorrhage into the brain. Had been excessive alcoholic. Demented, helpless, memory zero.

Shortly after entrance had another stroke, right hemiplegia, and died. Organic brain disease.

B. No. 20550. A brother of the above. Widower, 71 on entrance January 29, 1913. Showed typical senile dementia. No evidence of organic brain disease. Died of diphtheria February 28, 1913.

Representing second generation. There was a son of A, who was nervous, irritable, and married a neurotic woman whose mother is insane at 40. They were the parents of:



FAMILY NO. 15

C. *Representing third generation.* No. 22235. Female, single, 29 years old on entrance November 13, 1915. Oldest of six children who are said to be well. One brother alcoholic.

Did well in school. Nervous; had athyroidectomy. At 24 irritable, walked much, agitated, suicidal. Now in the hospital. Accused father of cruelty. No hallucinations; no other delusions. In hospital, apparently normal. Diagnosis, psychopathic inferior.

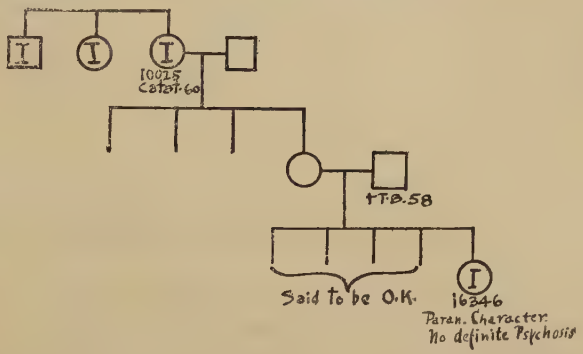
Summary. In the first generation, arteriosclerosis and senile dementia on the paternal side. (See chart.) On the maternal side, dementia praecox. The intervening second generation showed paternally, an "irritable person;" maternally, a "nervous person." In the third generation, hyperthyroidism with no marked mental disease. In other words in this family the two sides have been

"tainted," and yet the third generation shows only a psychopathic inferiority with hyperthyroidism. The rest of the members are said to be well.

FAMILY GROUP NO. 16

A. *Representing first generation.* No. 10025. Female, married, 60 years old on entrance May 16, 1877. Onset six months. Thinks there is a mob in the cellar coming to kill her. Is suicidal, apprehensive, mute, wakeful. Refuses food. Stands in one place continually. Resistive. Died suddenly of heart disease. Diagnosis, Spät Catatonic. (Late Catatonia, probably a form of dementia praecox.)

Two of her fraternity said to be insane. She was deserted by her husband because of her temper.



FAMILY NO. 16

One of her daughters married a man of average family. Their issue was B and two other daughters and two sons. All save B are said to be well.

B. *Representing third generation.* No. 16346. Female, married, 37 on entrance April 4, 1904. Always nervous. Three children well. For 11 years irritable. Quarreled continually with husband and children. Left him several times. Attempted suicide.

In hospital, nothing was found save the ideas against husband, which did not reach the status of insane delusions. Was considered only a paranoid character. Discharged May 30, 1904. Husband reports that condition has not changed. Diagnosis, paranoid personality.

Summary. The first generation, a character defect throughout life and then a late catatonic excitement. In the second generation, much tuberculosis. Third generation, mostly normal, one paranoid character.

FAMILY GROUP NO. 17

A. *Representing first generation.* No. 521. Male, 53 years old on entrance March 7, 1856.

In Worcester State Hospital twice. First attack at 25; insane by spells, noisy, boisterous, inquisitive, happy. Recovered in two months.

The second admission at 55, recovered.

In Taunton State Hospital, three attacks similar to above; one of distinct depression, the other two maniacal in type. He died at the age of 83 in 1886, cerebral hemorrhage. Psychosis, apparently manic-depressive of many short attacks. Never demented, never hallucinated. Mate unknown.

One brother had a daughter whose son represents the third generation, therefore A's grandnephew.

B. *Representing third generation.* No. 16033. Male, 41 years old on entrance August 7, 1907. A typical dementia praecox, paranoid type.

His father, however, not related to A, is insane and under guardianship. Therefore, it is probable that the psychosis in the grandnephew is related to the psychosis in the father rather than to that of the granduncle.

FAMILY GROUP NO. 18

A. *Representing first generation.* No. 9781. Female, widow, 59 years old on entrance April 30, 1886.

The physician's certificate, first attack, states: Acute mania, noisy, violent, and dangerous.

In hospital, "utterly demented and incoherent." Emotionally apathetic. Refused food, stomach tube used; became excited and died of diarrhoea September 10, 1886.

The next generation said to be normal.

B. *Representing third generation.* No. 19015. Male, single, 21 years old on entrance December 5, 1909. At 17 began to lose ground as worker. Believed he had heart, brain, and stomach disease, would be paralyzed. Seclusive and depressed. Gave up work at 19. Felt that he had lost manhood; was introspective, indifferent, attempted suicide. Discharged November 19, 1910. Probably incipient dementia praecox.

Of this family little can be said since the history is meager.

FAMILY GROUP NO. 19

A. *Representing first generation.* No. 47. Male, widower, 75 years old on entrance April 4, 1854. Had been insane at different times when young.

Entered the Worcester State Hospital at 64. Onset at 60. Melancholy. Occasionally noisy and troublesome. Appetite poor. In Worcester State Hospital usually stood in the corner with occasional excitement, but generally apathetic. In excitement, talkative and destructive.

September, 1848, Taunton State Hospital. Silly and happy, but usually takes no interest in environment. Better in 1854.

In Taunton State Hospital apathy, alternating with excitement and childishness. Died at 78 of fever.

B. *Representing third generation.* No. 10535. Grandson of A. Male, 38 years old on entrance March 29, 1889. Melancholy, deluded, suicidal, depressed. Heard his shopmates talking about him and criticizing everything he ever did. People want to drive him out of town. Said to have recovered entirely.

His mother, daughter of A, was melancholy. A maternal aunt was insane. A brother and sister died of tuberculosis.

Records are meager.

Probably no anticipation, some disease of manic type running throughout the three generations.

In the following three cases very little is known of the intervening generations and it is superfluous to give other than the following data:

FAMILY GROUP NO. 20

A. *Representing first generation.* No. 634. Male, 81 years old on entrance. Senile dementia.

B. *Representing third generation.* No. 4135. Grandson of A, 29 years old on entrance. Epileptic and feeble-minded.

FAMILY GROUP NO. 21

A. *Representing first generation.* No. 498. Female, married, 64 years old on entrance. Unclassified psychosis with melancholia.

B. *Representing third generation.* No. 20736. Granddaughter of A. Widow, 59 years old on entrance. Involution depression. In hospital at present time, unchanged.

FAMILY GROUP NO. 22

A. *Representing first generation.* No. 11423. Male, 68 years old. Senile melancholia.

B. *Representing third generation.* No. 17330. Granddaughter of A, 34 years old. Psychopathic with tuberculosis.

Two other families with three generations in the hospital are not here recorded, because in one general paralysis entered largely; in the other, very indirect lines are represented.

The following two generation (direct line) families have been selected from the large number at my disposal. *The reason for selection in each case has been not the type of mental disease presented in parent and descendant but expediency.* Many of the cases have

had one or more of the members in the hospital at the present time or within the memory of the present staff. Many of the cases have been selected because the records concerned were better than the average and, furthermore, a reason for selection in still other cases is that it has been possible to obtain an excellent family history. It is, therefore, to be again emphasized that these cases have not been selected to prove a point or to disprove one. Where haphazard selection has not ruled, the reason for selection has been expediency only.

I have divided these cases into groups according to the type of disease in the parent. It has not been possible to follow any exact classification in so doing and, therefore, I have designated certain of these groups, not according to the Kraepelinian or any other classification, but according to some leading character manifested in the psychosis. The reason for grouping the cases according to disease in the parent has been simply to ascertain, for example, the lines of descent, if there are any, in the psychoses; that is to say, to discover whether or not manic-depressive are followed by manic-depressive or dementia praecox, or what not. This has been the definite line of attack in the consideration of these cases.

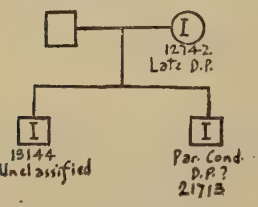
Group A

Paranoid type of psychosis in parent

Family group no. 23

A. *Representing first generation.* No. 12743. Female, married, 59 years old on entrance October 5, 1895. Onset at 57.

Father said to be "nervous," two sisters died of tuberculosis. A paternal aunt had shock.



Family No. 23

At 57, this patient fancied the family was about to be killed. Had headache, was sleepless, had hallucinations of hearing. Kept lights burning all over the house. Had marked delusions of persecution directed indiscriminately. Ideas of grandeur slight.

In 1896, apathetic.

In 1898, no change mentally. Died in 1906 with but very slight dementia. (No more dementia than might be expected at her age.) Diagnosis, paranoid condition.

B. *Representing second generation.* No. 13144. Son of A. Single, 33 years old on entrance September, 1896. Always difficult person to manage. Had to

leave home. Developed delusion he was to be married, invited everybody. Was grandiose but tractable. Concealed delusions in 1898 and was discharged.

Re-entered, No. 16096. Had the same delusion that he was to be married to a girl of the town. Was now exhilarated, happy, showed a true flight of ideas. Said he had any amount of money. Showed motor excitement and restlessness, euphoria, flight of ideas, boastfulness.

In 1907 he was transferred to the Medfield State Hospital where he is at the present time. Condition has remained practically the same.

In 1916, the blood and spinal fluid were examined with the result that the Wassermann was found positive in both. Diagnosis, was thought to be a typical manic-depressive insanity. The syphilitic infection may account for part of the condition, and therefore, I leave his case unclassified.

C. *Representing second generation.* No. 21782. Male, single, 55 years old on entrance December 24, 1914. Always seclusive. At 54 heard people talking against him. His sisters were against him. He became ugly and morose. Sat apart from others and would not eat with them.

In the hospital, no hallucinations; no memory defect; somewhat apathetic. Quiet, agreeable, worked well, no dementia. Entertained delusions of persecution against sisters; of rather coherent type. Died of tuberculosis February 3, 1915. Diagnosis, paranoid condition, possibly true paranoia.

FAMILY GROUP NO. 24

In this case we have three generations with hospital records, two of whom only have been at this hospital. Since, however, the records from the other hospital are excellent, all three generations will be given with some detail.

A. *Representing first generation.* Male, entered Brattleboro Retreat at 65, January 16, 1902. Onset one year before. No alcoholism. Had hallucinations of hearing and marked delusions of persecution. Said his wife was crazy and untrue to him. The neighbors were down on him and persecuted him in subtle ways. Delusions of reference marked. Threatened his neighbors.

In hospital, continued to entertain delusions of persecution which, however, were not well formulated or developed. Hallucinations of hearing; no dementia. Died October 30, 1907. Diagnosis of that hospital, paranoid.

B. *Representing second generation.* No. 20974. Female, single, 52 years old on entrance October 10, 1913. First attack at the age of 21, in Brattleboro Retreat—mania. Motor excitement, talkative, hallucinations of hearing. Discharged in six months, recovered.

Second attack at 35. Diagnosis of Brattleboro Retreat, manic-depressive with suicidal tendencies. Had hallucinations of hearing. Thought father and mother conspired to kill her illegitimate child. Recovered in six months.

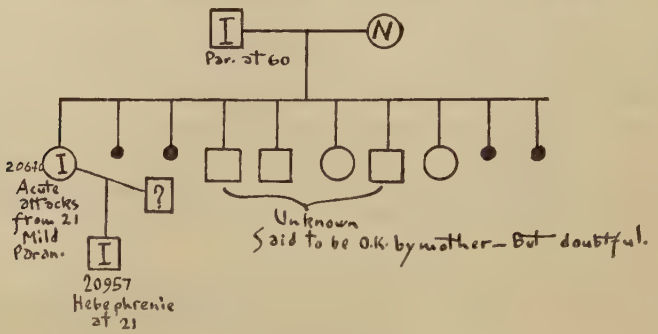
Third attack at 40. Had some excitement with hallucinations of hearing. Recovered.

Fourth attack at 41. Excited, confused, incoherent. Marked motor excitement, hallucinations of hearing. Discharged in six months.

Fifth attack at 42. Similar condition. Recovered perfectly.

Sixth attack, six months later. Said to have recovered.

At 51 the character of the disease seems to have changed. She entered the Boston Psychopathic Hospital October 28, 1912, and was then seen by the present writer. Marked somatic delusions. A snake is in her bowels. She has seen the snake at times. Irritable, but not noisy or violent. General emotional tone rather apathetic than otherwise. No dementia. Perfect grasp on surroundings. From time to time she had remissions when she no longer saw the snake. Developed the idea that she was spiritually influenced by some unseen powers. Had delusions of reference, especially directed against her neighbors. Believed she was being punished for her bad deeds.



FAMILY NO. 24

In Taunton State Hospital was a good worker, seclusive, apathetic rather than depressed. Entertained marked somatic delusions. Had hallucinations of sight and hearing. Showed no distinct dementia. Delusions of influence quite prominent. Was discharged to her mother, unimproved. Diagnosis of Taunton and Psychopathic hospitals, paranoid dementia praecox. It is probable that the attacks diagnosed as manic-depressive in Brattleboro Retreat should rather be considered as catatonic outbursts.

C. *Representing third generation.* No. 20951. Son of B, grandson of A. Male, 21 years old on entrance September 25, 1913. His case may be dismissed as being that of a typical apathetic dementia praecox, with delusions of persecution, hallucinations of hearing, and sexual ideas. He rapidly became deteriorated and was transferred to Grafton State Hospital April 6, 1915.

Summary. In this family with three direct generations represented there appeared first a senile paranoid state, then a probable case of dementia praecox, and last unqualified dementia praecox. The main change has been earlier onset of insanity with earlier dementia.

FAMILY GROUP NO. 25

A. *Representing first generation.* No. 12296. Male, 57 years old on entrance August 27, 1884. In the hospital at the present time. Onset at 49. Was irritable and suspicious. Neighbors persecuted him because of political differences. They stand outside of his window and make remarks about his genitalia. Women, in order to compromise him and ruin his political chances, solicit intercourse.

In hospital, coherent, no memory defect. Ideas directed against the democratic party and the Catholic Church. (He is a democrat and a Catholic.) Hallucinations of hearing influence his condition frequently. Very violent at times. Delusions faded out. The hallucinations disappeared. A mild dementia accountable by his extreme old age (at present 90) appeared. He worked every day until feeble. Still believes he is put here because, while dressing himself one day, enemies looking through the window saw his genitalia and spread the slander that he had exposed himself. Paranoid condition.

B. *Representing second generation.* No. 18632. Female, married, 39 years old on entrance March 26, 1909. Onset many years. She represented the 15th pregnancy of her mother and had two living brothers, both of them said to be normal. Was always jealous and irritable. Married at the age of 30. Domestic difficulties of high grade followed. She was continually quarreling with husband, had a bad temper and unreasonable jealousy. Destroyed household goods whenever crossed. Threatened suicide, and occasionally was violent towards her husband. Had a cancer of the breast which was operated upon.

In the hospital, no psychosis could be made out. She was pleasant, coherent, told a connected story, had no hallucinations and no delusions, unless the mild ideas of jealousy directed against husband could be so interpreted. She was discharged in a month with a diagnosis of psychopathic personality with paranoid trend.

Summary. In the two generations here described, the first showed at the involution period a paranoid psychosis, with hallucinations and absurd delusions. The daughter, always peculiar, developed no definite psychosis, but had a strongly paranoid and suspicious personality.

FAMILY GROUP NO. 26

A. *Representing first generation.* No. 18104. Female, married, 57 years old on entrance January 18, 1908. Discharged August 1, 1908.

Readmitted, No. 19256, at the age of 53, and is still in the hospital. Onset at the age of 50. Has ideas of persecution directed against the neighbors. Ideas of reference marked. Constant hallucinations of hearing. Outbursts of excitement with motor restlessness and destructiveness. Claims to have "double hearing." Noisy and talkative.

In hospital, similar condition with moderate dementia. Diagnosis of hospital, paranoid dementia praecox. Husband alcoholic.

B. *Representing second generation.* No. 22187. Daughter of A. Married, 33 years old on entrance October 6, 1915. Onset at 23. Suspicious and restless. Received messages by wireless telling her of impending destruction. Saw the devil kissing her child. Became very religious. Had delusions of reference and became very unstable emotionally.

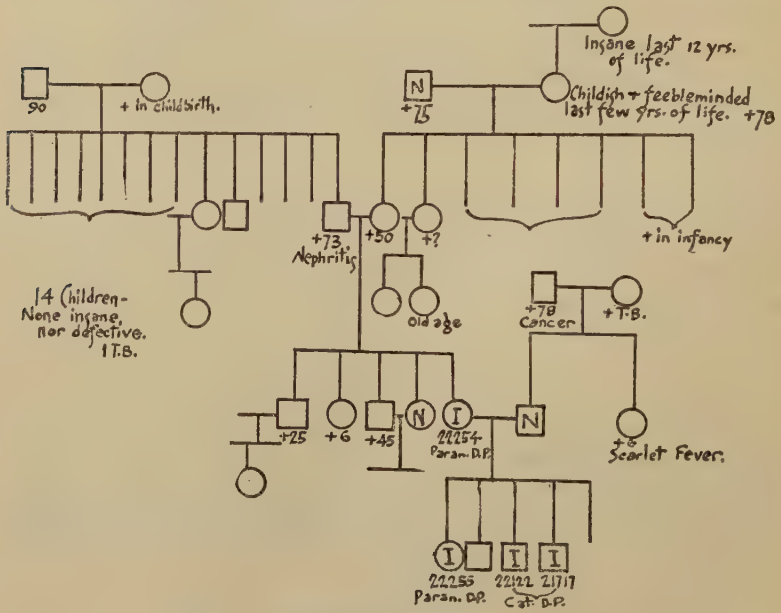
In the hospital has developed the delusion that she is worked on by vibrator and electricity for some vile purpose of the doctors. Hallucinations are marked. Generally apathetic. Diagnosis, paranoid dementia praecox.

Has one brother who is epileptic. Two brothers died at birth or just after. Two brothers said to be normal.

Summary. Paranoid psychosis in first generation, onset in involution period. Similar psychosis in second generation, onset at 23. In fraternity of second generation, normality, feeble-mindedness, and paranoid dementia praecox.

FAMILY GROUP NO. 27

A. *Representing first generation.* No. 22254. Female, married, 47 years old on entrance November 22, 1915. Said to have been bright as a girl. Was a teacher. Married at 20. Had four children, three of whom are now in hospital. Husband is said to be normal, but left family one year ago.



FAMILY NO. 27

Daughter, who is now in hospital, told her that men were pursuing her. She believed this and elaborated the delusion. Hobgoblins were employed by enemies to watch her and her daughter. Gases were squirted into the house. Poisonous electric currents were used against them. Her brother was trying to get rid of her property. She and her daughter threw their furniture out of the window to rid it of the poisons thrown on it.

In the hospital, memory excellent. Quick, smart, and apt in retort. Delusions are coherent and center around property. No hallucinations in evidence in hospital, but she is believed to be evading. Has developed delusions against one of the doctors, otherwise is friendly. Is a very good worker. Diagnosis, paranoid condition.

B. *Representing second generation.* (Daughter referred to above.) No. 22255. Single, 25 years old on entrance November 22, 1915. Onset at 21. Was also a teacher, but did poorly and was not so bright as the mother. Believed that men were following her dressed in women's clothes. Had hallucinations and delusions practically the same as mother.

In hospital, memory is good. Elated at times, silly, and occasionally lachrymose. Very poor retention of school knowledge. Much more marked hallucinations and more silliness than mother shows. Apathy improved somewhat on stay in hospital.

C. and D. (Twin sons of A.) *Representing second generation.* Nos. 21717 and 22315. Single, 20 years old. Similar psychosis. Irrelevant, incoherent, noisy, and quarrelsome at times. Generally mute, apathetic, and attitudinizing. Delusions of persecution very vague, some delusions of grandeur, some silliness and dementia. Stereotypy. Cyanosis of face and hands.

Summary. Four patients, representing two generations—a mother, daughter, and two sons. The mother and daughter share paranoid ideas, characterized on the mother's side by marked coherence and less hallucinations than on the daughter's. The mother shows more intelligence and greater natural mental endowment than does the daughter. The family history of mother is entirely negative. On the maternal side the grandmother of A (and therefore the great grandmother of the other members of this group) was "insane" the last 12 years of her life with senile dementia. In the next generation the mother of A was childish in old age. Of the brothers and sisters of A, none seems to have been abnormal though one was alcoholic.

FAMILY GROUP NO. 28

A. *Representing first generation.* No. 14547. Female, widow, 58 years old on entrance September 5, 1899. Onset at 48. Has marked delusions of persecution especially directed against the daughter and concerning property. She is very bitter and abusive verbally.

In hospital, excited and emotional. Memory good. No hallucinations or other delusions than those directed against the daughter, which are entirely without foundation. Was discharged November 3, 1899.

Re-entered April 12, 1900. Delusions very prominent and coherent, and still center around property. No dementia at the age of 64 when she was discharged to Medfield State Hospital. Last report from Medfield states: Not demented, still entertains delusions of persecution against daughter. Good worker. Diagnosis, paranoia, possibly paranoia vera.

B. *Representing second generation.* No. 16265. Daughter of A. Married, 32 years old on entrance January 6, 1904. Became insane directly after the birth of fourth child. Temperature between 103 and 104. Had acute nephritis. Generally stuporous. At other times marked religious delusions and hallucinations. Entertained delusions of reference. Believed herself hypnotized and influenced. Died one month after entrance, during which time temperature remained high and general condition resembled that of delirium. Diagnosis, toxic psychosis.

FAMILY GROUP NO. 29

This family has been described at length by Dr. C. T. McGaffin (American Journal of Insanity, lxviii, no. 2, 1911, p. 262), as a manic-depressive family. With this familial diagnosis I am unable to agree for reasons that will appear. It may be added that the hospital records do not so diagnose the respective cases. Two of the members of the family are still in the hospital and all the survivors, sane and insane, have been examined.

Family history . A negroid family with Indian and white intermixture. The mother of the first patient, according to Dr. McGaffin, has been insane with a short attack. The father, according to all reports, though a successful man, was overbearing and proud, and ordered his wife and children around as if they were inferior creatures. While I have not been able to obtain any history of the insanity of the mother, it seems very certain the father was a peculiarly paranoid type, and all of his grandchildren, especially mentioned this fact. Of these two persons were born 12 children, 9 of whom lived to adult life.

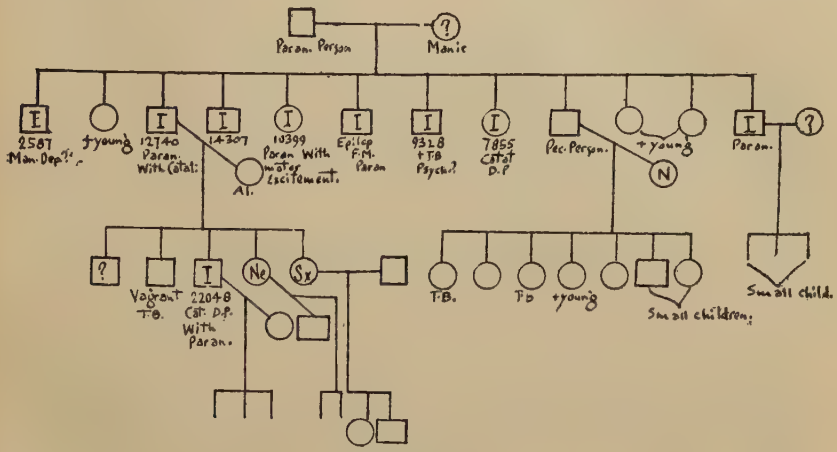
A. *Representing first generation.* No. 6522. Single, 24 years old on first admission. Four subsequent admissions. In interim of six years had three commitments to Danvers and to Taunton State Hospital. Had two admissions to a New York hospital. The last admission was to Taunton at the age of 35.

Each attack was characterized by excitement, talkativeness, grandiose ideas concerning ability as a detective, motor unrest, and quick recovery. After the last attack he remained for 20 years in the hospital in a normal condition, useful, quiet, likable, and under full liberty. He died of cardiac disease.

The records are scanty in this man's case, but the diagnosis of manic-depressive insanity is warranted. From my point of view this is the only case in the family in which this diagnosis can be seriously entertained.

B. *Representing first generation.* (No. 3 of Dr. McGaffin's series.) No. 12740. Male, married, 35 years old on entrance October 7, 1895. Had an attack at 32 after father's death, recovered. This attack was marked by excitement and strong delusions of importance. Believes he is a great detective and persecuted by other detectives because of his exposures of corruption in the city government. These delusions he retained for some time (one year) after the motor excitement had entirely disappeared. Then "recovery" ensued. Was discharged October 1, 1897.

Recommitted February 15, 1898, discharged March 1, 1904. Record unsatisfactory.



FAMILY NO. 29

Recommitted September 16, 1907, 65 years old. At present in hospital. Mute for months. Had marked catatonic traits, cerea flexibilities, negativism, distinct and decided religious delusions. Has seen Jesus Christ and holds communion with Him. Has marked mannerisms of speech and of expression. These mannerisms distinctly related to hallucinations of hearing being commands from God. Never depressed. At times cataleptic. Frequently shows marked catatonic traits. Always arrogant and suspicious.

At present, no dementia. Memory O. K. At times shows what seems to be a flight of ideas, *but is never distractible.* Generally suspicious and grandiose. Marked hallucinations. Frequently shuts his eyes and holds communion with Christ. Sometimes declares that he is the reincarnation of Christ.

The patient married an alcoholic negress. Had five children. The first is unknown. The second is a vagrant. The third is in the Taunton State Hospital at the present time (will be described later). The fourth is a female, ner-

vous, otherwise not remarkable. The fifth is a sex offender, has had two illegitimate children.

C. *Representing first generation.* No. 14307. Male, single, 54 years old on entrance August 26, 1899. Had his first attack at 34 and recovered. Four attacks of stupor in interim between this and the attack which brought him to Taunton State Hospital.

By occupation he was an inventor. Had always been difficult to get along with. Suspicious and proud. At the onset he believed he was being played upon by unseen batteries. Talked much about mental telegraphy, ate little, slept little, became exceedingly noisy.

On entrance to hospital declared he was Jesus Christ. Very haughty. Had "imperative delusions." Frequently took fixed attitudes and maintained them for a long time. Had quite active hallucinations of sight and hearing. At times disoriented. Claimed to be on a trip to earth for the second time doing his "Father's" business on earth. There was never any flight of ideas or distractibility. Never any change in condition. He died of tuberculosis in hospital. Diagnosis, paranoid condition.

D. *Representing first generation.* No. 10399. Female, single, 39 years old on entrance July 17, 1888. Had an illegitimate child.

Psychosis was marked by excitement, irrelevant and incoherent speech. Great restlessness of short duration. In interim between these attacks much motor and mental excitement; was exceedingly deluded. Had delusions of persecution and of a grandiose nature. She claimed to be a natural medium; owned the hospital; said that Great Britain and the United States were in difficulty over her family and her property. Had auditory and visual hallucinations. Showed many erotic manifestations. Never demented.

In 1893 was discharged to Worcester State Hospital where she remained until her death. The diagnosis there was dementia praecox, paranoid. The diagnosis in Taunton State Hospital was dementia praecox, paranoid.

Between this patient and the next one of her generation who entered the hospital was a brother who was an epileptic, feeble-minded, and was in the school for feeble-minded at Waverly where he showed peculiarities. He was seclusive, decorated himself, collected things. Was boastful and suspicious. He seems to have had a combination of conditions.

E. *Representing first generation.* No. 9238. Male, single, 35 years old on entrance August 5, 1884. The records are scanty. "Psychosis is characterized by ungrounded fear and apprehension of evil. Sometimes his mind will be much occupied with and excited on religious matters."

During his stay in the hospital was quiet but deluded. Had fixed delusions. These gradually faded out, but he remained easily aroused and was either very elated or very sad. Said to have recovered in May, 1886. Possibly a manic-depressive insanity, but records are too scanty to allow of any diagnosis. Died in 1903 of pulmonary tuberculosis.

F. *Representing first generation.* Nos. 9855 and 10462. Female, 32 years old on first entrance July 10, 1886. Was transferred to the almshouse September 25, 1886.

Was readmitted from the almshouse October 1, 1898, and discharged to Worcester State Hospital November 20, 1898. She was always incoherent, muttering, talking, resistive, occasionally very noisy and excited. Generally takes no interest whatever in environment. For 13 years, or until her death of tuberculosis in 1899 was unchanged. Generally speaking she was demented and deluded, with short periods of excitement. Undoubtedly a dementia praecox.

There were three more members of this generation; one, a male, who was never in the hospital, was always peculiar, suspicious, had grandiose ideas, and was continually in trouble. Was bright. He married and had six children. Three died of tuberculosis, one died of cholera infantum, and two are said to be normal.

Another male, peculiar, claims to be a doctor with divine power and went about giving treatment until arrest. Was always grandiose, quarrelsome, and seclusive. A female died young.

G. *Representing second generation.* No. 22048. A son of B in this series. Now in the hospital. Married, 47 years old on entrance July 6, 1915. Early life, always difficult person to get along with; ugly, quarrelsome, alcoholic. Within the last year developed marked religious delusions and became exceedingly excited. Had hallucinations of sight and hearing, these mainly of a religious nature. He claims to be the reincarnation of Christ on earth and is directed by Christ in all his actions. God talks to him. Takes fixed attitudes during which he is in communion with God. Exceedingly negativistic, very frequently has long continued mutism; very offensive in his air of superiority and at times very quarrelsome. Never any flight of ideas, not at all distractible. Apparently a typical catatonic dementia praecox.

Summary of cases. (See chart.) The father of the persons representing the first generation was, by all accounts, a man whose character may be summed up in the word, paranoid. He believed himself superior to most people with whom he came in contact. He showed marked egotism and had no consideration for the rights of others. One of his grandchildren declares that he died insane.

The mother, according to Dr. McGaffin's informants, had short periods of excitement and depression. My informants do not seem to know of this.

In the first generation, following these people, there were 12 members. Three died young, 5 became inmates of this hospital, 1 was an inmate of the Waverly School for Feeble-minded, and 3 were decidedly peculiar, and in the cases of 2 of these it may be said that they were insane without characterizing further the type of insanity. Of the 5 in this hospital, 2 present psychoses whose character cannot be determined from the records. In the case of the other 3 the records to my mind conclusively point to paranoid dementia prae-

cox. The descendant of 1 of these entered the hospital and has a psychosis that in almost every detail reproduces that of his father—the same religious ideas, the same tendency to catatonic attitudes, the same love of pompous and unmeaning phrases, and the same offensive air of superiority are found in each. The father shows no sign of dementia, though he is now an old man, and neither does the son. Aside from the psychoses presented in the second generation, we find vagrants, nervous individuals, and sex offenders. There is a good deal of tuberculosis in the descendants of these people. Whether or not to attribute these other conditions, that is, vagrancy, sex delinquency, and tuberculosis, to the "hereditary insanity" is a problem difficult to solve. It is an easy matter to say that a relationship exists, and yet these forms of deviation are very common and especially in a more or less mongrel stock, such as is represented by this family.

Group B

In the following cases the parent's condition was diagnosed as dementia praecox. The type of psychosis in the descendant will be detailed so far as is necessary to establish the diagnosis:

FAMILY GROUP NO. 30

A. *Representing first generation.* No. 15529. Female, married, 43 years old on entrance July 10, 1902. Onset one year ago, after influenza.

Maternal grandfather insane in Italy.

Patient is mute, attitudinizes, apprehensive, and restless. Has hallucinations of sight and hearing, negativistic. Later improved slightly, but was destructive. Was discharged November 21, 1904, with a diagnosis of dementia praecox, catatonic. Was slightly improved. Has been home since (1916). He has never been well, does no work, is demented.

B. *Representing second generation.* No. 22266. Daughter, single, 18 years old on entrance December 7, 1915.

In hospital at present. Has had "epileptic"[3] attacks foryears. Has always been nervous and seclusive, restless of late. Has marked hallucinations of hearing and delusions of reference. In the hospital is disoriented, confused, believes that the Jews and Portuguese persecuted her. (Unsystematized delusions of persecution.) Answers are irrelevant. Negativism is marked. Diagnosis, dementia praecox.

Summary. A grandfather insane, type unknown. The first hospital generation, dementia praecox at 40, catatonic onset. Daughter,

[3] Kraepelin mentions the frequency of epileptic attacks in dementia praecox.

dementia praecox at 19, with epilepsy. There are two brothers and one sister in the second generation. Other members are said to be normal.

FAMILY GROUP NO. 31

A. *Representing first generation.* No. 9995. Male, married, 37 years old on entrance January 30, 1887. Markedly excited at times, is threatening, seclusive, mute and resistive. Gradually recovered. Speaks no English. Was discharged, much improved, October 11, 1888.

Subsequent history (1916) shows that patient has never been really well. Has done but little work and is demented. A dementia praecox with catatonic onset.

B. *Representing second generation.* No. 21068. Female, married, 30 years old on entrance December 14, 1913. Has had epileptic attacks for years. Has been run down for years. One of her children is feeble-minded at Wrentham. She has had one miscarriage, one still birth, one feeble-minded child and two said to be well. Always a very difficult person to manage. Ran away from home. Recently developed delusions of unfaithfulness against the husband. Has delusions of reference directed against neighbors. Broke a window of home, took money, sold the furniture, and left.

In hospital, memory good; no hallucinations; no other delusions elicited save those directed against husband. Was discharged pregnant to husband January 3, 1914. Diagnosis, psychopathic personality with paranoid trend. Has epilepsy. Question of dementia praecox.

FAMILY GROUP NO. 32

A. *Representing first generation.* No. 13596. Female, married, 50 years old on entrance March 1, 1897. 46 at onset. Sleepless; believes herself pregnant and cannot be convinced to the contrary though years pass by and she is not delivered. Haunted by spirits.

In hospital has systematized delusions of persecution directed toward certain people who live in her community who send out spirits to trouble people. Has hallucinations of sight and hearing. Is aggressive and vile tempered. No dementia. Died of tuberculosis February 14, 1904, after seven years' residence in the hospital. Diagnosis, paranoid condition, probably dementia praecox. Married a negro who is said to have been well. There are three children. Of these, one is

B. *Representing second generation.* No. 16446. Female, single, 23 years old on entrance June 15, 1904. In hospital at present time. Onset at 22. Poor in school. Hallucinations of sight and hearing, and mild and indefinite delusions of persecution and reference. Very apathetic. Rapidly became demented.

Summary. Disease is worse in the descendant, and earlier.

FAMILY GROUP NO. 33

A. *Representing first generation.* Nos. 8272 and 10466. Male, 30 years old on entrance in 1881. Intemperate. Has delusions of persecution, is sullen and uncommunicative. Improved after first stay in the hospital and was discharged.

Re-entered at the age of 38 in 1889. Had delusions of poisoning, reference, and was very irritable. Became mute, negativistic, seclusive, and demented. Is in the hospital at the present time. Diagnosis, dementia praecox, complicated by alcohol.

B. *Representing second generation.* No. 17672. Son of A. Single, 32 years old on entrance March 5, 1907. In hospital at present time. Onset at 30. Has hallucinations of sight and hearing. Generally apathetic. Has occasional sudden and violent outbursts. Marked somatic delusions; evasive. General gradual mental decline. Is good worker under supervision, but takes no interest in environment. Diagnosis, dementia praecox.

Summary. In this family we have a similar psychosis at a similar age.

FAMILY GROUP NO. 34

A. *Representing first generation.* No. 8382. Male, married, 63 years old on entrance August 11, 1881. Onset at 53. Preliminary history not obtained.

In hospital, sluggish, apathetic, demented. Exceedingly obstinate and negativistic. Occasionally violent; generally indifferent. Vague delusions of persecution. Dementia praecox.

B. *Representing first generation.* No. 8901. Sister of A, 56 years old on entrance June 16, 1883. Insane at 41. "Generally mild," occasional violent outbursts.

In hospital, hallucinations of hearing, delusions of influence by spirits. Apathetic, indifferent, grew demented. May 1, 1896, discharged to Medfield State Hospital. Dementia praecox.

C. *Representing second generation.* Nos. 16593 and 19363. Daughter of A, 38 years old on first entrance October 16, 1904. Always poor mental capacity. At 36 began to grow suspicious. Became very sensitive. Had delusions of reference and jealousy. Became seclusive and indifferent.

In 1913, deeply demented at 47 years of age, sullen, resistive, quarrelsome, hallucinated. Died of mitral regurgitation in 1914. Autopsied by present writer. Dementia praecox.

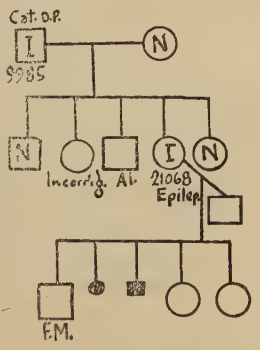
D. No. 17328. Cousin of A and B. Question of whether paternal or maternal relationship. Female, single, 81 years old on entrance June, 1906. Had tremor for 20 years. Was never "just right" mentally. Troublesome for many years. Lately hallucinations of sight and hearing. Delusions of persecution and untidiness. Died August 17, 1907. Question of senile dementia or old dementia praecox.

In this family, the only noteworthy phenomenon is the appearance of anticipation. The psychoses are in general similar.

FAMILY GROUP NO. 35

A. *Representing first generation.* No. 21232. Male, married, 64 years old on entrance March 6, 1914. Insane for many years. No definite history obtained. Indefinite delusions of persecution. Demented; hallucinated; apathetic. Good worker. In hospital at present. Diagnosis of dementia praecox, paranoid symptoms. His wife is at Medfield State Hospital with dementia praecox. Onset at 25. One daughter is there.

B. *Representing second generation.* No. 21195. 40 years old on entrance February 18, 1914. Very typical hebephrenic dementia praecox. Markedly demented. In hospital at the present time.



FAMILY NO. 35

Summary. The first generation, both male and female, are dementia praecox. The descendants show the same disease at not dissimilar ages.

FAMILY GROUP NO. 36

A. *Representing first generation.* No. 14556. Female, married, 38 years old on entrance April 21, 1900. Typical demented, hallucinated dementia praecox, bench type. Marked sexual delusions. Was sent to Medford State Hospital January 5, 1904.

B. *Representing second generation.* No. 17178. Daughter of A. Single, 15 years old January 24, 1906. History shows absolute lack of sexual morals. Cohabited with any man who desired her, totally shameless. Untrustworthy in statements of any kind. Inclined to be boastful and lazy. Very careless in personal appearance; has very poor mentality. Unable to be promoted in school. Diagnosis: "moral imbecile."

Summary. In this case the transition in the second generation is towards "moral imbecility" from dementia praecox in the first generation.

FAMILY GROUP NO. 37

A. *Representing first generation.* No. 16330. Female, married, 37 years old on entrance March 17, 1914. In hospital at present. Onset at 30. Was married at the age of 20. Two children. One illegitimate child after husband's death. Always tended to be depressed and seclusive.

In hospital, excited, hallucinated, talkative, irritable. Simple and childish. Does no work. Likes to take off her clothes and expose herself.

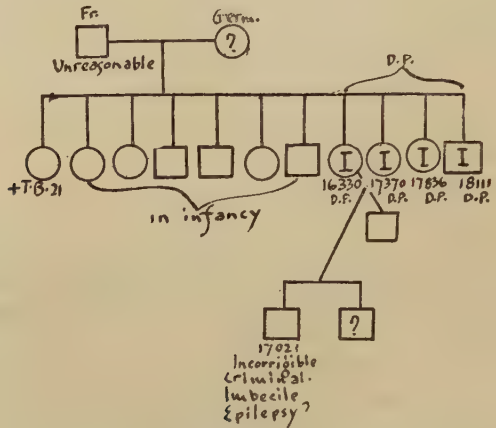
In 1910, deeply demented, apathetic.

In 1916, apathetic, bench type of dementia praecox.

B. *Representing first generation.* No. 17370. Sister of A. Female, 44 years old on entrance June 20, 1906. In hospital at present. Condition practically the same as sister except less excitable.

C. *Representing first generation.* No. 17370. Sister of A. Female, 44 years old on entrance October 18, 1906. Similar condition. Died of lobar pneumonia November 2, 1907.

D. *Representing first generation.* No. 17371. Brother of foregoing. 30 years old on entrance June 20, 1906. Second admission at 37. In hospital at the present time. Has very marked delusions of persecution of absurd character, religious delusions, and hallucinations of sight and hearing. Marked mannerisms, dementia, apathy, and seclusiveness. Considered typical dementia praecox.



Family No. 37

E. *Representing second generation.* No. 19120. Son of A. 13 years old on entrance September 30, 1908. Has been in orphan asylum. Is absolutely incorrigible. Lies and steals, and is a mischief maker of worst kind.

In hospital, superficial brightness, no exact knowledge of anything. Boastful—sticks pins into other patients. Lies continually. Steals whatever he can regardless of its usefulness or its value. Had several fainting attacks which in many respects resembled petit-mal. Can neither read nor write, despite the fact that an attempt has been made to teach him. Diagnosis, imbecile with perhaps epilepsy.

Summary. In the first generation the psychoses are practically the same. In the second generation the one individual represented is an imbecile with marked lack of moral sense.

FAMILY GROUP NO. 38

A. *Representing first generation.* No. 17228. Female, married, 45 years old on entrance March, 1906. Onset probably within last few years. Received a severe burn a month before. Became disoriented, silly, untidy, noisy. Low-grade, feeble, and demented. Died in June, 1906. The mental condition antedated the severe burn by a few years. Was regarded as dementia praecox.

B. *Representing second generation.* Nos. 13796 and 14383. Daughter of A. At present in hospital, first entrance June 2, 1898, at the age of 20. Very typical hebephrenic dementia praecox. Markedly demented, apathetic, hallucinations. Sits on bench most of the time.

Summary. The early onset of the disease in the daughter is the only noteworthy feature.

FAMILY GROUP NO. 39

A. *Representing first generation.* No. 10581. Female, married, 35 years old on entrance June 15, 1889. 33 years old at onset. With third child had "pain in the head;" refused to eat, was violent and shouted.

In the hospital, dull, apathetic, monosyllabic in answers. Had hallucinations of hearing, though deaf. Became indolent, seclusive, and irritable. Sent on trial December 9, 1889, against advice.

Readmitted May 20, 1891, No. 11107, condition worse. Vague delusions of persecution in addition to above symptoms. Developed a hard, dry cough. Died of tuberculosis July 14, 1892. Dementia praecox.

B. *Representing first generation.* No. 16439. Sister of A. 35 years old on entrance January 10, 1904. In hospital at present. At 30 began to develop well-marked delusions of persecution which became full-fledged on entrance. Had hallucinations of sight and hearing, delusions of reference, poisoning, and persecution. Became slovenly, dirty, irritable and negativistic. Later developed sexual delusions. Had hallucinations of smell. Became very sarcastic. At present decidedly demented.

C. *Representing second generation.* No. 20543. Daughter of A, niece of B, 24 years old on entrance January 23, 1913.

Ran away from home at 16, became a prostitute in New York. Became a morphine habitué. On entrance to the hospital was mute, dull and apathetic. Late, tore clothes, became untidy, and smiled in a silly manner. Grew demented and sat on bench with silly expression on face.

Summary. First generation, onset at 30, paranoid dementia praecox. Second generation, onset at 24, catatonic onset. Moderate anticipation.

FAMILY GROUP NO. 40

A. *Representing first generation.* No. 12651. Female, married, 33 years old on entrance July 3, 1895. Onset at 31.

Father alcoholic. A half-sister on the paternal side was insane a short time.

The patient entered after a pregnancy. Was excited, sleepless, exalted; believed that she would rule the world and had billions of dollars.

Later, became exceedingly erratic; had hallucinations of hearing. Was violent, noisy, and disturbed, agitated. Apathy profound towards last of stay in hospital. Was sent to Medfield March 6, 1897. Died of typhoid fever at 36. Dementia praecox, paranoid type.

Her husband was a normal man. Had five children. One died of cholera infantum, one died unknown, three in hospital.

B. *Representing second generation.* No. 19341. Son of A. 19 years old on entrance September, 1910. 17 at onset. Poor mentality. Masturbates. A vagrant for some years.

In hospital, silly, irrelevant, incoherent, vague fear. Had marked splitting up of speech ("word salad"). Apathetic and thoroughly demented in 1916 at the age of 25.

C. *Representing second generation.* No. 19432. Brother of B. 23 years old on entrance October 26, 1910. In hospital at present.

Condition similar to brother's.

D. *Representing second generation.* No. 19594. Sister of B. 23 years old on entrance March 20, 1911. Always considered feeble-minded. Mental condition not different from that of brother.

Summary. In this family the anticipation occurs despite the normal mate. Undoubtedly the feeble-mindedness is very early dementia praecox.

FAMILY GROUP NO. 41

A. *Representing first generation.* No. 15694. Female, married, 28 years old on entrance Nov. 11, 1902.

Has had five attacks, each in hospitals. At first improved between attacks. Attacks lasted usually about three months. Later, no real remission. Each attack characterized by excitement, incoherence, talkativeness, violence and destructiveness. In the third attack, distinct delusions commenced to appear. These concerned a neighbor and her mother. Delusions of persecution became prominent. Negativism decided. Untidy. Changeable emotional tone. Hallucinations of hearing. Transferred to Medfield State Hospital in 1906. Died of pulmonary tuberculosis. Diagnosis in this hospital and in Medfield, dementia praecox. This diagnosis rests on the later attacks, for the first two were diagnosed and seemed like manic. The appearance of a continual psychosis with only slight remissions, ideas of persecution, negativism, and hallucinations substantiates the diagnosis of dementia praecox.

B. *Representing second generation.* No. 19242. Daughter of A. Single, 14 years old on entrance June 9, 1910. Onset at 12. A friend committed suicide. She developed the delusion that the friend was chasing her and that everybody was dead. Had hallucinations of sight and hearing to substantiate this belief. Recovered.

The attack which brought her to the hospital commenced three months before entrance. Hallucinations of sight and hearing concerning her dead mother. Attitudinized. Improved and was discharged September, 1910.

Returned at 16, No. 20124. At the present time in hospital. Deluded, hallucinated, and demented. Dementia praecox.

Summary. The disease in both mother and daughter is characterized by an onset in attacks. No essential difference between the disease in mother and daughter, except that it started earlier in the daughter.

FAMILY GROUP NO. 42

A. *Representing first generation.* No. 7091. Male, married, 46 years old on entrance June 13, 1879. Onset six months before.

Patient became quickly demented. Sat around with bowed head. Untidy, incoherent, mute. Acted like the bench type of dementia praecox. Diagnosis, dementia praecox. Discharged to almshouse April 14, 1886.

B. *Representing second generation.* No. 21485. Daughter of A. Female, single, 48 years old on entrance July 6, 1914. Now in the hospital. Always considered feeble-minded. Lately developed vague hallucinations and delusions of reference. Moderate ideas of grandeur. Childish and apathetic. Sits around the ward all day. Diagnosis, feeble-mindedness and dementia praecox.

Summary. In the second generation the patient's mental condition is complicated by feeble-mindedness, probably of the praecox type.

FAMILY GROUP NO. 43

A. *Representing first generation.* No. 18173. Male, married, 45 years old on entrance March 23, 1908. Maternal grandfather nervous at times. Maternal grandmother died at 70. Father alive at 70, infirm. Mother not remarkable at 63. One sister nervous and one died in childbirth.

Patient has never been quick to learn. Sensitive. Married at 23; had 13 children, of whom two died, one miscarriage, ten alive. "God first spoke to him at the age of 32. Has heard His voice ever since. Has heard other voices, but does not know the identity of the speakers." Recent memory is rather poor. He quarreled with his wife. Had nervous spells at times, and because of his threatening attitude was sent to the hospital.

In this hospital his memory improved. The confusion which marked his earlier stay disappeared. He quickly came back to a somewhat feeble-minded normal and was discharged from the hospital June 24, 1908. Diagnosis, based on the history that he had been hallucinated for many years, that he had periods of confusion and excitement and that he seemed to be growing mildly demented, is dementia praecox.

B. *Representing first generation.* No. 10626. Sister of A. 23 years old on entrance August 19, 1889. This is the second attack.

Onset of the first attack at 19. She was violent, destructive, and profane. Had very decided delusions of persecution directed against husband.

In hospital, noisy, excited, incoherent, refused food. Was decidedly untidy, had hallucinations of hearing, and delusions of poisoning.

In April, 1890, obstinate, delusions of persecution and reference, irritable. hallucinations of sight and hearing. Very seclusive. Discharged November 26, 1980, not improved. Diagnosis, dementia praecox, paranoid form.

C. *Representing second generation.* No. 18711. Daughter of A. Single, 22 years old on entrance June 1, 1909. Auditory and visual hallucinations at times for years. Epileptic attacks for years. Believes she has a beautiful voice and will become a famous singer. Very decidedly feeble-minded. No active psychosis observed during stay in the hospital. Was discharged August 25, 1909. Diagnosis, feeble-minded plus epilepsy, possibly dementia praecox.

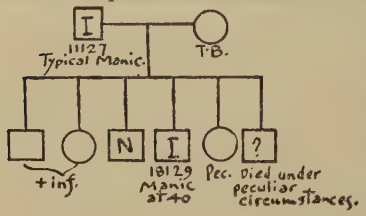
Summary. In the first generation we have two individuals, a male who (while able to carry on his life outside the hospital) showed backward mentality and a light psychosis with paranoid ideas and fleeting hallucinations. His sister is decidedly a paranoid dementia praecox. His daughter, in the second generation, has been feeble-minded since birth and at times has had auditory and visual hallucinations.

Group C

The following group of cases are best considered under the heading of manic-depressive insanity, although as will be evident when the cases are analyzed some of them, strictly speaking, do not belong here. Here I am following, however, merely the precedent set by most of the writers, who have utilized a heading, "The manic melancholic group" under which they include certain depressions which apparently belong outside of the Kraepelinian manic-depressive group:

FAMILY GROUP NO. 44

A. *Representing first generation.* No. 11127, male, married. This patient had 19 hospital commitments from the 16th to the 65th year. All attacks were alike. Began with mild depression, from this the patient passed quickly to facetiousness, talkativeness, excitement, flight of ideas, and motor restlessness. There was a quick recovery in each case. No hallucinations, no paranoid delusions, no dementia. He died at home, aged 66, in 1892. His wife died of tuberculosis at the age of 40.



FAMILY NO. 44

B. *Representing second generation.* No. 18129. Son of A. Married, 40 years old on entrance February 2, 1908. Onset three months before. He kept his wife awake at night talking about religion. Began to investigate the forms of religion in order to choose the right one; began to be very busy at home, making plans on a large scale. Became restless at night. Finally took to bed and was sent here.

In hospital, quiet, evasive, coherent, though vague about religion; not elated nor particularly depressed. Spoke about the brotherhood and the sisterhood of man, the fatherhood and the motherhood of God, had to love all men, etc. No hallucinations. No delusions other than these. Recovered quickly and discharged May 2, 1908.

Summary. The manic-depressive disease in the ancestor is typical. The disease in descendant has changed somewhat, but undoubtedly belongs to the manic-depressive group. The onset of the first attack in the descendant was at much older age than that in the ancestor.

FAMILY GROUP NO. 45

A. *Representing first generation.* Had four commitments to this hospital. Nos. 10233, 10964, 11076, and 11854; age, 24, 27, 31, and 34. Female, married, The attacks are similar. Occurred at the end of each lactation. Became melancholy, retarded, suicidal. Each attack commenced with restlessness, apprehension, and depression. Believed that God wanted her to die. Her children were coming to harm; everything was wrong in the world. Never hallucinated. No memory defect. Recovery took place in each attack in about two months. Depressed phase of manic-depressive insanity.

B. *Representing second generation.* No. 20889. Son of A. Single, 31 years old on entrance August 21, 1913. In hospital at present. Unquestionably a case of dementia praecox.

History of excessive masturbation. At present apathetic, catatonic symptoms at times, silly, confused, negativistic, markedly demented.

Summary. We have one manic-depressive followed by dementia praecox. The husband of A (father of B), is perhaps a trifle nervous, but is otherwise normal.

FAMILY GROUP NO. 46

A. *Representing first generation.* No. 13301. Female, married, 40 years old on entrance January 25, 1897. The onset was at 38. Sleepless, poor appetite, became suicidal, apprehensive, and confused.

In hospital was depressed, agitated, cries and moans, wrings her hands. Says she will never be better and cannot sleep. Occasionally shows marked excitement.

After a month in the hospital commenced to gain steadily. Recovered completely. Discharged May 26, 1897. She died five years afterwards *in*

childbirth. No mental symptoms. Some phases of this patient's case resemble involution melancholia, but the fact that the involution period had not yet taken place with her and that five years later she was pregnant ruled out this condition. I have classified her as agitated depression.

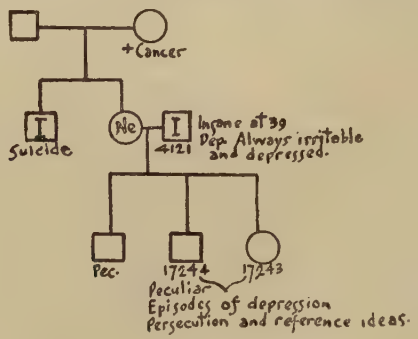
B. *Representing second generation.* No. 19506. Son of A. Single, 26 years old on entrance January 9, 1911. Has delusions of persecution and reference. Is suspicious and mute. Has marked somatic delusions and is apathetic.

During stay in hospital had hallucinations of sight and hearing, was suspicious of his environment; at all times seclusive and deluded. Died of pulmonary tuberculosis in 1913. Dementia praecox, paranoid form.

Summary. In this family there is a transition in the psychotic type—first generation—manic-depressive insanity, agitated depression; a dementia praecox, paranoid, in the second generation.

FAMILY GROUP NO. 47

A. *Representing first generation.* No. 4121. Male, married, 41 years old on entrance January 14, 1871. First attack at 39. Became worried about busi-



FAMILY NO. 47

ness. Feared that he was to lose his property. Became exceedingly depressed. Tried suicide. Beat his head against the wall. No hallucinations; no dementia. Recovered March 7, 1871, discharged. Is said to have been nervous and at times unbalanced the rest of life. Whether a diagnosis of manic-depressive can be made in this case is problematic. Possibly we are dealing with an upset of a different nature in a psychopathic person. At no time was there retardation and the depression seemed related to the affairs of his life.

B. *Representing second generation.* No. 17243. Daughter of A. Married, 17 years old on entrance March 27, 1906. Always did poorly in school. Was very overbearing and envious. Had not lived with husband for some time, Sent to Butler Insane Hospital in 1905. Had delusions of unworthiness, was

restless, retarded. Mood generally one of discontent rather than depression, according to records. Attention poor. No improvement. At home had delusions of financial persecution on the part of her husband. Continually and easily disturbed. Believed she would be put in jail. The housekeeper, she thought, had caused her mother's death. Was untidy. Combed her hair continually. Together with her brother, who was sent to this hospital, she tried suicide and was sent to Taunton.

In Taunton remained unchanged. Was in a disturbed state, suspicious, and anxious. Believed that she was to be put in jail and that her husband and the housekeeper were conspiring against her. Very poor memory, very poor judgment in most affairs. Continually at odds with the people around her. Discharged in six months against advice, unimproved.

C. *Representing second generation.* No. 17244. Brother of B. Male, 42 years old on entrance March 27, 1906 (same day as sister). Always peculiar. His psychosis in general may be said to be similar to the sister. Suspicious, hostile to environment, depressed, easily agitated, never happy or contented. Incapable of doing any work or of keeping his attention on anything for any length of time. Tried suicide with sister. In hospital behaved well, worked well, was always seclusive and suspicious.

Summary. Undoubtedly there is mental disease on both sides of the family. The mother of B and C, wife of A, was nervous, and had a brother who was insane and committed suicide. The father's psychosis (A) cannot be easily classified, but belongs to a group in which character defect is prominent throughout life and in which episodes of excitement and depression with a substratum of suspiciousness occurred under stress. In the second generation there were three individuals, two of whom came to the hospital. One was peculiar. The other two cannot easily be classified. There was a basis of low mentality with a paranoid character trend. The attempt at suicide, the paranoid attitude towards the environment, the discontent rather than depression bring the conditions nearer to dementia praecox than to manic, but no classification seems well to cover the case unless it is that of psychopathic personality with psychosis.

FAMILY GROUP NO. 48

A. *Representing first generation.* No. 18599. Male, widower, 80 years old on entrance February 25, 1909. First insane attack. Always successful; fairly prosperous. Shortly after wife died became suicidal, inclined to cry, was much depressed, very decidedly retarded. Memory was fair. He is said to have heard his dead wife calling, "Come." After he had been in the hospital for a few months he improved, became cheerful, contented, and recovered. He remained in the hospital, working and acting in every way normally until

1913, when he had diarrhoea and then a cerebral hemorrhage from which he died March 19, 1913.

B. *Representing second generation.* No. 20163. Son of A. Married, 30 years old on entrance June 1, 1912. He had a short attack following mother's death four years before. One year before commitment, hypochondriacal ideas. Believed he was going blind; his testicles were diseased; stayed in bed all day and was decidedly depressed. Occasionally irritable.

In the hospital, no hallucinations, no definite delusions, markedly retarded, impaired flow of thought; memory good, orientation good. Recovered and was discharged. Diagnosis, manic-depressive insanity, depressed phase.

C. *Representing second generation.* No. 13645. Male, married, 28 years old on entrance December 4, 1897. Sudden onset. Feared people were coming to kill him. Was retarded. Tried to injure his wife and himself. Incoherent and occasionally hallucinated.

In the hospital decidedly retarded, expressionless face, depressed; mutters to himself. No interest in environment. No hallucinations. After six months went on to complete recovery. Discharged December 18, 1898. It is reported that he had another attack later on from which he recovered.

Summary. In the first generation a depressed attack at the age of 80 which was suicidal and went on to complete recovery. Both of the sons were suicidal, depressed, and recovered. Marked anticipation.

FAMILY GROUP NO. 49

A. *Representing first generation.* No. 11131. Male, married, 68 years old on entrance June 21, 1891. Coherent, depressed, suicidal, violent especially at night. Memory for recent events poor. The attacks started with great depression, confusion, and hopelessness in regard to health. Thought he was going to die. Much retarded. No hallucinations or other delusions. In two months he recovered from mental symptoms entirely. Remained in the hospital until November 21, 1892, when he died of valvular heart disease. The attack seems to have been a simple depression. His wife died of cancer of the breast.

B. *Representing second generation.* No. 3138. Son of A. Single, 16 years old on entrance June 25, 1867. First depression at 15.

In hospital, is excited, thinks he is Christ. Is violent and restless. Later became depressed and stayed in bed. Was discharged in 1868.

He had a similar attack in 1872, No. 4686, aged 20. Later in California Hospital for the Insane. It is impossible to make a definite diagnosis from the scanty facts adduced. *Primary delusional insanity* was the diagnosis of the clinicians at that time. This corresponded in general to the diagnosis of dementia praecox at present.

C. *Representing second generation.* No. 16781. Son of A. Male, married, 48 years old on entrance March 1, 1905. Had a depression at 33. At 46 became tired, lacked ambition, became seclusive and depressed. Very irritable

if disturbed. No hallucinations. Said he was a disgrace, a tramp, no good. The men in the shop called him names.

In the hospital, grew irritable and sarcastic. Very evasive in his replies. Not so much depressed as hostile. Rather indifferent. No dementia. Stayed in bed continually. Died June 11, 1906, of hypostatic pneumonia.

Summary. In the ancestor we have a senile melancholia from which recovery followed. There were three sons. Two had depression as a leading symptom but with more sinister symptoms, that is, symptoms which point toward a chronic psychosis. In the one, the psychosis took on a grandiose nature in that he believed he was Christ. In the other, there was the development of a hostile attitude towards the environment. There was an earlier onset of mental disease. One brother not in this hospital had "epilepsy."

FAMILY GROUP NO. 50

A. *Representing first generation.* No. 9140. Male, married, 50 years old on entrance June 26, 1884. Father died of shock, mother of dropsy.

The patient was insane for a short period at 21 and recovered. Now threatens suicide, is depressed. Says that the stomach and bowels are decayed, food does no good. Retarded. Discharged August 22, 1884, recovered.

Re-entered, No. 11035. Was well until December, 1890. Then had a similar attack and discharged January 21, 1891, recovered. He died in 1911. Had occasional "blue days," but said to be well up until the time of death. Diagnosis, manic-depressive insanity, depressed phase.

B. *Representing second generation.* No. 17496. Daughter of A. Single, 24 years old on entrance October 3, 1906.

Mother peculiar. Her half-sister was insane and her daughter, a half-sister of B, was also insane and in this hospital with dementia praecox.

This patient became depressed, cried, was restless, hypochondriacal, occasional hallucinations, but seldom and indefinite. Very emotional, seclusive. Never demented, always depressed and uneasy. Apprehensive of something to happen. Gets on well in the hospital, but does badly at home. Is a boarding-out patient and never has given trouble. At present, aged 34, while there is no marked depression and no active mental symptoms she is continually worried and alarmed.

Summary. In the first generation, we have a rather typical manic-depressive. In the second generation, two insane stocks enter. On the mother's side there was evidently dementia praecox both in her half-sister and in another daughter by another husband. B seems to have had a condition that may better be called chronic depression than to be fitted into either of the manic-depressive or the dementia praecox groups, and which almost seems a blend between dementia praecox and manic-depressive.

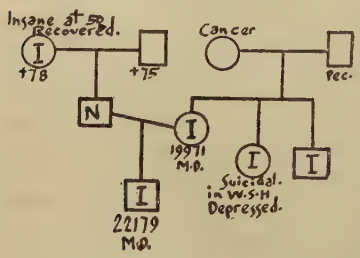
FAMILY GROUP NO. 51

A. *Representing first generation.* No. 15874. Female, married, 44 years old on entrance March 12, 1902. Had two previous attacks, one at 16, when she was excited, exhilarated, and mischievous. Lasted one year. One at the age of 40 lasted two months. This attack commenced with depression. She became dejected, slept poorly, ate nothing, and resisted all efforts at feeding her. Died of inanition a week after admission. She had a brother who died insane.

B. *Representing second generation.* No. 21757. Son of A. Single, 22 years old on entrance December 12, 1914, patient was a heavy drinker. Developed hallucinations of hearing. Became markedly apprehensive, excited, and incoherent. Voices threatened him continuously. Quickly recovered and was discharged January 13, 1915. Diagnosis of acute alcoholic hallucinosis was made.

FAMILY GROUP NO. 52

A. *Representing first generation.* No. 19971. Female, married, 42 years old on entrance June 29, 1912.



FAMILY NO. 52

Mother died of cancer. She has a sister at Westboro—manic-depressive insanity. Brother committed suicide, probably insane. The first attack at 37. Brooded, depressed. Recovered in two weeks.

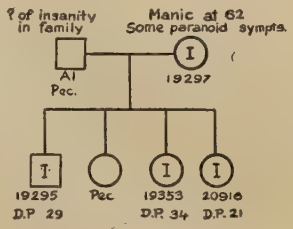
Four months before admission this attack—depressed, attempted suicide. Said bowels would never move, pleased to be killed. Retarded. Showed marked confusion. Recovered July 31, 1912. Went home and has been well since. Simple depression of manic-depressive insanity.

B. *Representing second generation.* No. 22179. Son of A. Single, 17 years old on entrance October, 1915. Is in hospital at present. Pressure of activity. Shows marked flight of ideas. Recovered within three months. Manic-depressive insanity, excited phase.

The family history here is extremely interesting. Of the ancestors of the father, who himself was normal, the mother was "insane" at 50 and recovered. On the maternal side, that is, on the side of A, her father was peculiar and the mother died of cancer. Her brother and sister both were insane so that there is a bilateral taint in so far as B is concerned. This, however, seems to have in no essential way changed the character of the psychosis. Anticipation is the only phenomenon here of any significance. It may be noted that while the mother's psychosis was essentially a depression, the son's was essentially manic, which tends to demonstrate the biological validity of the Kraepelinian union of the manic and depressed states.

FAMILY GROUP NO. 53

A. *Representing first generation.* No. 19297. Female, widow, 62 years old on entrance July 14, 1915. For year had been worrying over two children in hospital. Lately began to laugh, sing, swear, dance, and talk in vulgar manner. Showed extremely marked motor restlessness. Was talking continuously. Had flight of ideas. Was destructive. *Fears food has been tampered with.* Later, admitted active hallucinations of hearing. Was very quarrelsome. Occasional fleeting delusions of persecution. Improved steadily, discharged March 17, 1912, and well since. Hospital diagnosis was manic-depressive insanity, manic phase. This diagnosis is open to some doubt in the face of the very active hallucinations of hearing. However, there seems to have been nothing else in the case that might possibly make it one of catatonic excitement. She had two daughters and one son in this hospital.



FAMILY NO. 53

B. *Representing second generation.* No. 20919. Daughter of A. 21 years old on entrance September, 1913.

C. *Representing second generation.* No. 19295. Son of A. 29 years old on entrance July, 1910.

D. *Representing second generation.* No. 19353. Daughter of A. 34 years old on entrance December 19, 1910.

These patients are still in the hospital. All of them show very marked paranoid dementia praecox with dementia, hallucinations, and violent, aggressive conduct at times. Generally, the emotional tone is one of extreme apathy and indifference. They are as typical cases of this condition as can be found. A factor which must be considered in B's case is the paternal inheritance, that is, on the side of the husband of the patient, A. He was an alcoholic and had a niece in an asylum in England. Further than that we have no evidence as to the type of psychosis presented by the niece or as to the general character of the man. Anticipation is a marked phenomenon in this case. Hallucinations and ideas of poisoning in the mother's psychosis were associated with symptoms otherwise almost typically manic. The same ideas of poisoning appear in the descendants.

FAMILY GROUP NO. 54

A. *Representing first generation.* No. 3079. Male, single, 22 years old on entrance August 13, 1887.

Following typhoid the patient became violent, attacked parents. Expressed the wish that he were dead. Very destructive. Recovered. February 13, 1868.

Readmitted April 26, 1894. No. 12160. Married, 48 years old. Had been perfectly well in the interim. Following business trouble became sleepless.

Believed he had killed somebody and suspected that it was his own child. Very suicidal. Claimed to be sick all over. Markedly agitated. Pounded his head against the wall, disrobed, was destructive and confused. Had to be tube fed. Died December 6, 1894, without any change in condition.

Each of his attacks followed acute infection and it is possible that they are to be regarded as exhaustion psychosis rather than as manic-depressive insanity or any phase of it.

B. *Representing second generation.* No. 18606. Daughter of A. Single, 16 years old on entrance March 4, 1909. Precocious, peculiar. After measles, at the age of 13, was not herself. Refused to eat, had visual hallucinations. Apprehension was marked. Developed ideas of grandeur and made a partial recovery.

On entrance was excited, talkative, sang at times, feverishly alert. Expressed ideas of grandeur. Was euphoric. Claimed that Teddy Roosevelt was her father, this in an attempt to be humorous. Shameless in conduct and speech. No hallucinations noted. Recovered, March 24, 1910.

Readmitted, No. 19578. March 17, 1911. Following a fire became confused, depressed, agitated. Disconnected conversation, restless. No hallucinations. Recovered September 20, 1911. The diagnosis in her case made of manic-depressive insanity seems to be justified. Each attack, however, in so far as the records go, followed either an infection or some exciting occurrence, such as fire.

Summary. The daughter's psychosis occurred somewhat earlier than did the father's and the attacks were longer with less intermission. The family history shows very much mental disease on the parental side.

FAMILY GROUP NO. 55

A. *Representing first generation.* No. 1234. Female, married, 43 years old on entrance May 11, 1859. Thinks she has committed an unpardonable sin. Is a sinner above all others. Will be eternally damned. Does not know the nature of the sin. Very depressed, quiet. Other delusions whose character is not mentioned in the record. No hallucinations. Very markedly improved by August 6, 1859, when she was discharged. Never really well afterwards. "Had funny notions."

B. *Representing first generation.* No. 9180. Sister of above. Married, 73 years old on entrance July 18, 1884. Always peculiar. At times considered half crazy. Always hypochondriacal and suspicious. At 66 had a stroke. Since then violent, abusive, malicious in speech. Spreads slander. Absurdly suspicious and antagonistic. No hallucinations. Conduct disorder marked. No paralysis. Was discharged improved in a month.

C. *Representing second generation.* No. 15130. Son of A, nephew of B. Single, 40 years old on entrance August 22, 1901. In hospital at present. Father alive at 70. Paternal side negative. Only son of A. She had one brother and one nephew who were insane as well as sister.

Patient did well in school, always good-natured. Always extremely worrisome, self-deprecatory. Began to feel that his work was unsatisfactory. He believed that a man in the shop hypnotized him because he refused to buy a lottery ticket. At night this man would crawl along his window shade and then along the fence, howling at him like a cat. Later believed he was poisoned. Carried around samples of food for analysis.

In hospital, extremely apprehensive, clearly oriented. Convinced that one of the patients on the ward is another man, an acquaintance. Extremely suspicious.

In December, 1910, agitation increased because the influence was being played on him. People were reading his mind. Complained that murder, fire, etc., passed through it. Feels that patients on ward are distinctly inferring that he is an Odd Fellow, that he is a Mason, he drinks, has illegitimate children, etc. Fell into a catatonic state, assumed positions which he resisted changing. Remained catatonic and negativistic for months, then gradually recovered. By January 15, 1912, was improved. Since then has been in apparently normal condition. Works in the library of the hospital. Is zealous, overconscientious, has an absolute lack of self-confidence, but at the same time a marked childish egotism. Is eccentric. Became very stout and laid out a plan of exercises and diet which he followed religiously until he had reduced his weight 70 pounds. Has a marked and childish interest in the little things around him. Memory is good. A slight resemblance to Mr. Taft, the ex-president, has filled him with great joy and he likes to remark to strangers that people tell him he looks like Mr. Taft.

Summary. A was peculiar, had a short depression with typical ideas. Then a mental enfeeblement without any active symptoms. Classification of this cannot be made on the symptoms presented. A sister of this woman was peculiar. Was paranoid, a difficult person to get along with. This was increased by a stroke. A son of A, also peculiar, always with a feeling of inadequacy, had a psychosis preceded by marked feelings of inadequacy, of influence, and at its height characterized by catatonic symptoms. There was recovery to a childish, hypersensitive condition. In all three of these cases there is a fundamental character defect which in two of the members took the form of inadequacy; in one of the members, B, took the form of a paranoid attitude. In the last case the psychosis was diagnosed by all who studied it as catatonic dementia praecox and I feel that this is the correct diagnosis. In the case of the mother, A, the psychosis cannot be definitely classified although it was considered to be simple depression.

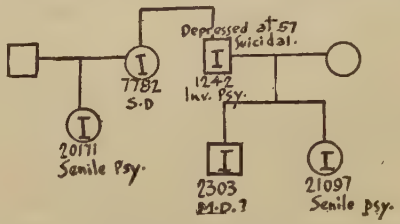
FAMILY GROUP NO. 56

A. *Representing first generation.* No. 1242. Male, married, 57 years old on admission May 24, 1859. Onset at 57, sudden. "Coming to want, no hope in life, all sorts of conflicts are coming, markedly agitated." Suicidal and succeeded in committing suicide in the hospital.

Brother was insane. A paternal uncle insane. The diagnosis of an involution type of depression seems justified. Whether or not manic-depressive can be considered is a question.

B. *Representing second generation.* No. 2303. Son of A. Single, 30 years old on entrance January 28, 1864. Hospital notes are extremely scanty. "He was flighty in his talk, noisy in conduct." Was discharged, recovered, August, 7, 1865. Died at 80. Said to have been normal throughout his life.

C. *Representing second generation.* No. 21097. Daughter of A. Married, 75 years old on entrance December 23, 1913. Always peculiar. Bright; generous. Her main peculiarity consisted in her talkativeness to strangers especially and she was shunned because of this peculiarity. For over 40 years she had been considered insane. Conduct, however, aside from this talkativeness had been



FAMILY NO. 56

exemplary. She lived with an older brother, became untidy, disorderly, and through some accident the brother was burned to death when the house took fire.

In the hospital, talkative, conversation extremely irrelevant, very fault-finding, quarrelsome. Memory intact. Apparently has occasional hallucinations of sight. There was present, however, no dementia. No real flight of ideas, but a marked logorrhea. Absolutely no insight into her condition. Took sick shortly after entrance. Died with unresolved pneumonia January 26, 1914. Personally autopsied. Diagnosis, psychopathic personality which merely deepened as life went on. Whether the talkativeness was part of a chronic manic state is of importance, but as she had been in the hospital less than a week when she became sick, this question cannot be answered.

D. *Representing first generation.* No. 7782. Sister of A. Married, 72 years old on entrance September 12, 1878. Records, which are full, show a typical senile dementia with loss of memory, restlessness at night, quarrelsomeness, and childish demeanor. Died Oct. 10, 1888.

E. *Representing second generation.* No. 20171. Daughter of D. Widow, 85 years old on entrance June 4, 1912. Undoubtedly insane for many years.

Conduct had been peculiar for over 30 years. Always extremely set in her opinions, seclusive, jealous, with condition that had for its origin unreasonable motives. Lately developed delusions of persecution against the relatives. Feared her food was poisoned. No dementia. Talkative. No hallucinations. Mood querulous. Died September, 1912, of pneumonia.

Summary. The two families here grouped in one present interesting facts. In the group first presented, the ancestor A, who came of a markedly insane family, had a psychosis which originated in the involution period and ran a rather typical course. A son had a psychosis from which he recovered. Nothing further can be stated concerning him except that he died in old age. A daughter, always peculiar because of her marked loquacity, drifted into an asylum in old age through social mishap and helplessness. The psychosis in her case seems to have been a mere development from her temperament. The ancestor of this group had a sister D, whose psychosis seems to have been purely senile dementia. Her daughter E, had a mental disease coming on in old age which seems to have been like her cousin's case, in that it represented merely a deepening of the loquacity and general suspicious attitude of her temperament. In the cases of C and E, the two cousins, the most striking phenomenon is the deepening of an originally peculiar temperament into a condition which becomes socially disharmonious and which results in incarceration into an insane hospital. This peculiarity of the psychoses which results in commitment in old age will be dealt with later.

FAMILY GROUP NO. 57

A. *Representing first generation.* No. 5264. Female, married, 29 years old on entrance August 26, 1873. Following childbirth patient began to talk, sing, and show extreme motor restlessness. Was very noisy, troublesome and destructive. Hallucinations not mentioned. No definite delusions. Was actively excited for two months and then quickly recovered. Discharged February 11, 1874. Complete recovery.

Readmitted at the age of 42, No. 9829. Menopause and death of sister given as cause. Was incoherent, talked continuously; confused, exalted, irrelevant conversation. Noisy, disoriented.

July 18, 1887, note says, "Quiet, very seclusive, depressed." Recovered April 5, 1888.

Two attacks are thus recorded, each following some definite event—the first marked by motor excitement and recovery, the second marked by motor excitement and later depression and recovery. Manic-depressive insanity is the most likely diagnosis.

B. *Representing first generation.* No. 4757. Sister of A. Married, 38 years old on entrance July 16, 1872. Insane since birth of last baby, six weeks ago. Had been insane after former childbirth.

In hospital, excited, mischievous, shameless, and troublesome. Talkative, destructive. Recovery complete November 15, 1872. No further attacks. Died in 1886.

C. *Representing second generation.* No. 19944. Son of A. Married, 45 years old on entrance December 30, 1911. Always of a mild, quiet and retiring disposition. His wife was ill three months and he took care of her night and day. On her recovery became sleepless, disturbed, tried to jump from window, became exceedingly talkative.

In the hospital, at first quiet and depressed. Heard sad voices. Later became markedly agitated. Said his bowels were gone. His nerves were gone. He must go to work at once to ward off impending poverty. Excited. Died of erysipelas without recovering from mental symptoms March 25, 1912.

In this family psychoses followed definite events. In the case of the two sisters, childbirth brought about motor excitement from which there was recovery. Their cases classified better as manic-depressive than under any other heading. The son of the first sister, also became insane following the strain of nursing a sick wife. His psychosis seems like an agitated depression with a possibility of an involution melancholia. Unfortunately for him and for the settlement of the question of whether or not he would have recovered from his psychosis, he died of erysipelas. It is probable that he would have recovered so that in general the same biological picture is presented in the cases of the three individuals.

In 1916, when this was written I was strongly under the impression that "real" psychoses after disturbing events, and in a cause-effect relationship to those events, were unusual. Since then my experience in the community as a practitioner has convinced me that exhaustion and sudden emotions, even great joy, can bring about mental states of severe type.

Group D

The following group of cases are those in which the parent suffered from a psychosis at or about the involution period of life. The type of mental disease cannot be exactly determined in some of the cases. Some undoubtedly present the syndrome of involution melancholia. Others are perhaps late dementia praecox and still others are to be classified as Spät Catatonie.

FAMILY GROUP NO. 58

A. *Representing first generation.* No. 11879. Female, married, 60 years old on entrance July 26, 1893. "Full of worms, can't eat—the insides are spoiled. Can pass no urine or feces." Later she became indecent, sleepless, violent, suicidal, generally depressed, and agitated. Masturbates openly. Still

later asks to have her head cut off. "The people around her are being killed and butchered. Why is she let live?" Very markedly suicidal. Became quieter, good ward worker, cheerful. Discharged December 2, 1896, recovered.

Case belongs to the group of diseases known as involution melancholia. There is a typical agitation, somatic and nihilistic delusions. Depression was very marked. Urstein describes such cases as Spät Catatonie and it is possible that they are best classed under this heading. The patient's father had asthma. A brother died of kidney disease. No mental disease known.

B. *Representing second generation.* (Three persons).—No. 11226. Son of A. Single, 33 years old on entrance September 19, 1891. Had been depressed for a month, became very religious. Felt that he had lost his chances of salvation, became noisy, excited and dangerous.

In the hospital refused food. Was intolerant of all clothing, destructive, incoherent, showed motor excitement and died of exhaustion in a week, October 4, 1891. Catatonic dementia praecox is the diagnosis made at the hospital, but this seems erroneous, and the diagnosis is more likely depressed phase, manic-depressive insanity.

C. No. 17007. Daughter of A. Single, 42 years old on entrance September 28, 1905. Second attack. Gradual onset six weeks. Had been melancholy, followed by active excitement. Became profane, obscene, incoherent, screamed and gesticulated. Had hallucinations.

In hospital, very resistive, markedly apprehensive, completely confused. Died in six days. Autopsy held, no cause of death disclosed.

D. No. 19912. Daughter of A. 34 years old on entrance April 30, 1910. The father of the patient had died of pneumonia and old age. We learn now that the mother, patient A, died in old age with no mental symptoms.

Patient had always been seclusive. A month before believed that the rest of the family did not love her. They thought she was in the way. She refused to eat except under persuasion. Became suicidal. Acquired a delusion that she had committed some great sin.

In hospital, was mute most of the time, picked her face and her nails. Very resistive. Died May 30, 1910, of erysipelas.

Summary. The diagnostic problem involved in these cases is great. We may conceive of an agitated form of manic-depressive as running through the entire family, starting in old age in the mother, starting earlier in the children, and in the cases of the children leading to death within a short time. We may also consider that the mother had involution melancholia and that the three children suffered from manic-depressive insanity; or a catatonic disease may be diagnosed in all four cases with recovery in the mother and death from exhaustion in the children. Whatever be the diagnosis, it seems to me clear that the type of mental disease in all four cases is the same, that in all there is a marked depression, a marked feeling of inadequacy (feeling of sin, of having been ruined), a marked agitation, and motor ex-

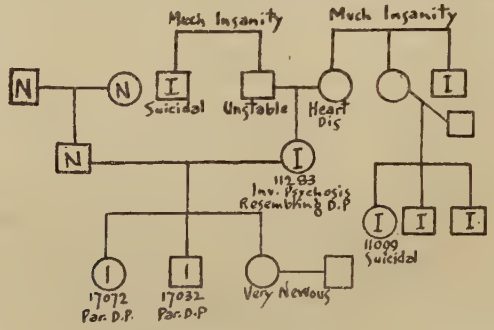
citement. Hallucinations and delusions, if present, were not prominent. In this group anticipation, that is, the earlier onset of the psychosis, becomes prominent in the second generation.

FAMILY GROUP NO. 59

A. *Representing first generation.* No. 11283. Female, married, 44 years old on entrance December 10, 1891.

Brother was insane. There was much mental disease on the paternal side as well as on the maternal side (see chart).

Patient mated with a normal man of normal stock. The first change occurred at 38. Became sleepless and had a marked failure in appetite. Developed hypochondriacal ideas. Has malignant disease which she has communicated to other and communicates it to everybody with whom she comes in contact. Refuses food. Has mild delusions of persecution.



FAMILY NO. 59

Hospital History. Somatic ideas persisted for years. Later, disappeared. Delusions of poisoning persisted for years, faded out. Became apathetic and without initiative. Died August 24, 1905, aged 62. Diagnosis, involution psychosis, possibly involution melancholia, possibly a dementia praecox type.

B. *Representing second generation.* No. 17032. Son of A. Single, 32 years old on entrance September 30, 1905. Very backward, unsuccessful, poor worker. Seclusive, shy, and suspicious. Generally depressed. Developed hallucinations of hearing, delusions of persecution, and sex delusions. No insight; orientation fair. At times aggressive. Discharged to Medfield State Hospital March 20, 1908. Diagnosis here and at Medfield, dementia praecox, paranoid form.

C. *Representing second generation.* No. 17072. Daughter of A. 28 years old on entrance October 26, 1905. Psychosis similar to brother's in its general features. Transferred to Medfield March 18, 1908. Diagnosis here and there, dementia praecox, paranoid.

D. No. 11009. A maternal first cousin of A. Female, married, 51 years old on entrance October 5, 1887. In the hospital at the present time. Onset at

28, following the birth of third child. Was depressed and later had elated periods. These followed in rapid succession throughout her stay in hospital. Occasional halucinations, not marked. In lucid intervals very pleasant and very fond of tracing her ancestry. Occasionally has confused period with fleeting and vague hallucinations of sight and hearing.

At the present time has a depression, is retarded, somewhat negativistic, and untidy. Very sociable, at other times no dementia. The diagnosis in this hospital is manic-depressive insanity. Diagnosis at McLean Hospital where she has been is manic-depressive insanity, circular form.

Summary. In this group we have a normal stock on the father's side uniting with an insane member of an insane stock. (See chart.) Involution psychosis in the first hospital generation followed much mental disease in her ancestors. In the second generation there were three individuals, two of whom reached the hospital, with paranoid dementia praecox. The third individual is said to be very nervous and peculiar, perhaps represents a starting point for a new group of "insane" persons. If we consider only the two hospital generations, there is anticipation. If we include the generation immediately preceding A, anticipation disappears as a prominent factor.

FAMILY GROUP NO. 60

A. *Representing first generation.* No. 13619. Male, widower, 55 years old July 31, 1897. "Everything wrong everywhere, a machine has turned the world upside down." Coherent about past life; quiet generally. Indefinitely and confusedly expressed philosophical ideas. Feels that he has a mission to change the world, and accosted strangers, asking for money to cleanse Taunton. Later, developed mild delusions of persecution and poisoning.

In 1898, depressed. Voices tell him he must soon die. Kneels and prays a good deal. Not retarded. Believes that Satan causes stagnation around his heart and his head. Quiet, apathetic. Transferred to Foxboro State Hospital August, 1905. Diagnosis—the apathy, general character of the delusions, the slow progress of the disease—is late dementia praecox. Involution psychosis must be considered.

B. *Representing second generation.* No. 12676. Daughter of A. Single, 25 years old on entrance March 7, 1907. Has always been seclusive and worrisome. Believed herself too good to mingle with others. Wanted to become a nun. Gradual onset of the disease. Brooded, became without initiative, had a marked feeling of inadequacy, wanted to die. Said that her stomach and lungs were gone. No one to help her. She cannot eat. No hallucinations, no dementia. Orientation good. *Very decidedly resistive and negativistic.* Discharged April 24, 1907, improved. Letter from home says she is well. Hospital diagnosis, manic-depressive insanity. With this diagnosis, I agree. While the two diseases in the two generations are dissimilar outwardly, yet

essentially the underlying ideas and the underlying feelings are that things are all wrong with the individual and with the world. That is to say, there is an inadequacy of the individual and a disarrangement of the environment. This inadequancy is transitory in the daughter, at least in its acute form. In the parent it goes on and is built up into further delusions and later is accompanied by hallucinations. Despite the fact that there are marked dissimilarities, fundamentally the psychoses are not different. Anticipation is present in that the psychosis occurs early in life in the daughter.

FAMILY GROUP NO. 61

A. *Representing first generation.* No. 13757. Female, 51 years old on entrance April 2, 1898. Patient is depressed, agitated, and restless. Believes that neighbors annoyed her. She misinterpreted their actions into persecutory conduct. Had hallucinations of hearing. Delusions of persecution are wide-spread, but not systematized.

Later, in the hospital, became agitated, depressed, markedly hallucinated. Improved slightly; discharged October 28, 1899.

Re-entered December 5, 1906, aged 53. Has been only moderately improved in the interim.

At present very melancholy, apprehensive, has poor memory, agitated. Rubs hands together continually and has no interest in the people around her. Became decidedly demented. Transferred to Medfield State Hospital. Diagnosis lies between involution melancholia and dementia praecox. The appearance of marked agitation and depression together with the characteristic wringing of the hands made a diagnosis of involution melancholia acceptable to the hospital staff. I am not satisfied that this is the correct diagnosis, but am in no position to debate the matter.

B. *Representing second generation.* No. 21564. Male, 33 years old on entrance September 11, 1914. One sister and one brother died of tuberculosis. Onset recent. Believes that the unions are against him and persecute him in many ways. Spirits bother him. Is stupid and confused. Later, became apathetic. Believed that the hospital was doped and the poison was being brought into his system. Had hallucinations of hearing. November 18, 1914, discharged against advice. Diagnosis dementia praecox, paranoid.

FAMILY GROUP NO. 62

A. *Representing first generation.* No. 13201. Female, 40 years old on entrance November 11, 1896. Onset very recent, within last week. Says husband sold his daughter's soul to the devil. Stands all day praying to drive this spirit from the daughter's soul. Speaks no English. Apprehensive, very markedly disturbed, and excited. Within a few days became tractable and quiet. Became cheerful and apparently normal. Remained normal and died of pneumonia at home at 63. Diagnosis, unclassified. Possibly catatonic excitement.

B. *Representing second generation.* No. 13202. Daughter of A. Single, 18 years old on entrance November 11, 1896. For four weeks an evil spirit had possessed her. "The Lord has married her to a young man."

Will not talk, at times was excited, sleepless, and noisy. Answering hallucinations continually. Had delusions of reference and persecution. Was active and destructive for weeks. Discharged, unimproved, in December, 1896. Went to Canada.

Re-entered, No. 14087, February 8, 1899. Still noisy and excited. Destructive, hallucinated, deeply demented. Was transferred to Medfield State Hospital December 4, 1899. Was there in 1916. Demented, occasionally excited, generally apathetic, and indifferent. Hallucinations of sight and hearing. Diagnosis, dementia praecox.

C. *Representing second generation.* No. 18634. Daughter of A. Married, 38 years old on entrance March 23, 1919. Onset sudden. The mother had recently died and patient became exceedingly noisy. Wanted everybody to pray for her, wished to become a saint in order to save her father and brother. Recovered within a week. Says she did it on purpose to make her husband and her father stop drinking and thus save the souls of the members of the family. During her psychosis she had visions of various kinds which were interpreted symbolically. Told how the Sacred Heart spoke to her and told her to do what she did. Discharged, completely recovered, April 24, 1909. No hallucinations or delusions. Good insight into past condition.

Summary. It is noted that the mother had the same delusion as did the daughter, that is, that her soul had been sold to the devil. To this idea she reacted with agitation and depression from which she recovered. The older daughter at the time of the mother's death, that is, following a definite emotional disturbance, had a sudden psychosis with hallucinations, delusions, agitation, and depression from which she also recovered quickly. The younger daughter had a psychosis marked by agitation, depression, and excitement, but with the added features of a typically excited dementia praecox from which she did not recover and which passed into the apathy, indifference, and dementia of dementia praecox.

Whether the psychoses in the mother and older daughter are to be considered as belonging to one type of mental disease and different from that in the younger daughter is a question. To my mind, they are merely lighter forms and are to be regarded as transitory catatonic states. There is present here anticipation and worsening of disease type.

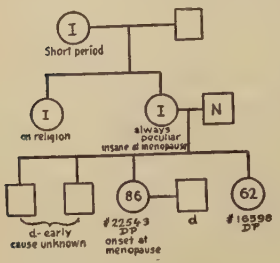
A son of C, the older daughter, died of convulsions, which has no great significance.

FAMILY GROUP NO. 63

A. *Representing first generation.* No. 17309. Female, widow, 54 years old on entrance May, 1906.

Rapid onset, markedly hallucinated, was so violent as to be inaccessible. Very indecent, very destructive, sang and screamed.

Later condition unchanged. Very resistive and noisy. No flight of ideas. Continually reacts to hallucinations. Transferred to Medfield and died there at 57. Diagnosis, catatonic dementia praecox, appearing in involution.



FAMILY NO. 63

B. *Representing second generation.* No. 17753. Son of A. Single, 16 years old on entrance April 20, 1907. Sudden onset. Became mute, resistive, refused food, stayed in bed, occasionally had attack of violent excitement. Generally apathetic, indifferent.

Recovered in four months. Never depressed, never showed manic symptoms. Discharged October 19, 1907. Said to be well in 1916. Married and has a family.

C. *Representing second generation.* No. 21680. Daughter of A. Married, 30 years old on entrance October 17, 1914. Said to have tuberculosis. Two children well. Was deserted by husband. Has delusions of reference of a fantastic type. Marked delusions of persecution and poisoning. Judgment very poor; for example, does not realize she is in an insane hospital. No insight. Incoherent.

Improved slowly in hospital. Discharged, not recovered, December 18, 1915. Diagnosis, paranoid dementia praecox.

Summary. In this group a catatonic excitement occurring at 54, without improvement leading to dementia and finally to death, was followed in the second generation by a short catatonic excitement at 16 in one member and a paranoid dementia praecox in a second member at 30. There is anticipation.

FAMILY GROUP NO. 64

A. *Representing first generation.* No. 14068. Male, married, 53 years old on entrance January 27, 1899. At 52 had a short attack of some ill-defined psychosis. At 53 suddenly developed ideas of poisoning. These lasted for a few days. He became excited, walked into the home of a stranger and asked for the hand of his daughter. Believed he was to marry this young girl. Was happy and elated, coherent. Acted as if under auditory hallucinations, was talkative. Became suddenly violent, attacked an attendant, was roughly handled and received injuries from which he died. Diagnosis not made. Acute mania of unclassified nature, perhaps due to alcohol.

B. *Representing second generation.* No. 21910. Daughter of A. Married, 26 years old on entrance November 13, 1915. In the hospital at present. Psychosis had its onset after last baby. Has had three children. Has delusions that blood was poisoned and something was growing inside of her. Her bowels are upside down. Hallucinations of sight prominent, also of hearing. Confused, apprehensive, distinctly hostile to persons around her.

Later, became negativistic and immodest. Continually hallucinated. At present is apathetic, indifferent, moderately demented, has hallucinations of sight and hearing, delusions of persecution and of a somatic nature. Diagnosis, dementia praecox.

She is one of 10 children, two of whom died early. No others insane.

Summary. In this family an unclassified maniacal attack is followed by dementia praecox at an early age.

FAMILY GROUP NO. 65

A. *Representing first generation.* No. 11331. Male, married, 42 years old on entrance January 25, 1892.

Father was at one time insane and a brother died of tuberculosis.

In the hospital, incoherent, talkative, hallucinations of hearing, marked confusion. No clear delusions. Emotional tone neither elated nor depressed. Rapidly cleared up and was discharged January 15, 1892.

Re-entered, No. 12656, 45 years old in August, 1895. Mute, acted silly. Was confused, excited. Ate but little, grew thin. Mental condition quickly recovered. Was discharged December 25, 1895. Said to have been well since. Apparently catatonic excitement, possibly manic-depressive insanity.

B. *Representing second generation.* No. 18697. Son of A. Single, 22 years old on entrance May 20, 1909. Inability to work for years, morose and disagreeable, egotistic, stubborn. Developed delusions of reference and persecution. In hospital, believed that he was influenced by electricity. Had hallucinations of sight and hearing. No distinct dementia. Sullen. Discharged, unchanged, May 10, 1914. Diagnosis of hospital, dementia praecox, paranoid.

FAMILY GROUP NO. 66

A. *Representing first generation.* No. 18999. Female, married, 54 years old on entrance December 10, 1909.

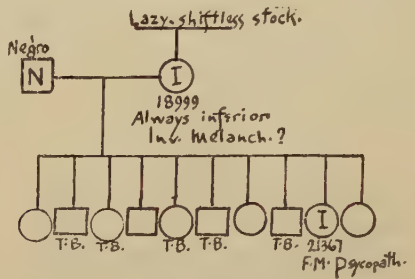
She comes of a shiftless, lazy family, generally despised in the community. At 18 was pregnant by a negro whom she married. Always slack and untidy. Some previous "nervous prostration," never insane.

In the hospital, marked apprehension, delusions her children are to be killed, that she is to be killed. Has hallucinations accordingly. Agitated. No memory defect. Orientation good.

Slightly improved after the first year. Discharged August, 1911, recovered. Died in 1912 of pneumonia. Diagnosis in hospital was manic-depressive insanity. This diagnosis is questionable to me because of the marked hallucinations, the lack of flight of ideas, the retardation. However, I have no diagnosis to set up against it.

B. *Representing second generation.* No. 21367. Daughter of A. Single, 21 years old on entrance May 24, 1914.

Of 10 brothers and sisters of this generation, five died of tuberculosis. Grief over an unfortunate love affair made her take poison. Stayed in bed, refused to work, was silly in her conduct.



Family No. 66

In hospital, no definite psychosis was made out. She was careless of attire, poor in judgment, and decidedly of low-grade mentality. Discharged August 12, 1914. Diagnosis, psychopathic inferiority.

Summary. In the ancestor an involution psychosis which may belong either to involution melancholia, or to dementia praecox from which the patient recovered. In the descendant feeble-mindedness with a mental condition of short duration marked by silliness and inertia. Diagnosis made by hospital staff, psychopathic inferiority.

Group E

The following seven cases have the common character that organic brain disease was diagnosed in the case of the ancestor. In the first three, the family history was unobtainable. In the next three, a good family history was obtainable, and in the seventh case other peculiarities entered which will be detailed in the case.

family group no. 67

A. *Representing first generation.* No. 19702. Male, married, 58 years old on entrance August 31, 1911.

Had right sided hemiplegia. Demented. Memory defect, disorientation, and confusion. Died March 15, 1913.

B. *Representing second generation.* No. 19766. Son of A. 28 years old on entrance in September, 1914. Depressed and restless. Wandered at night, occasionally excited.

In hospital, dull, apathetic, hallucinations of hearing, refuses to work. Poor grasp on surroundings. Gradually grew weaker. Diagnosis of tuberculosis was made. Discharged May 8, 1915, to die at home. Dementia praecox, hebephrenic.

FAMILY GROUP NO. 68

A. *Representing first generation.* No. 8581. Male married, 63 years old on entrance June 24, 1882.

Onset at 61 after apoplectic stroke. Violent at times and suicidal. Paralytic.

In hospital, demented, blind and deaf. Died November 24, 1882, after stroke.

B. *Representing second generation.* No. 9056. Son of A. Married, 35 years old on entrance December 14, 1883.

Had somatic delusions, food does not pass through him. The bowels are jammed. Has a holy spirit in his heart. Is incoherent, destructive, auditory hallucinations. Became demented and died of tuberculosis. Diagnosis, dementia praecox.

FAMILY GROUP NO. 69

A. *Representing first generation.* No. 19792. Male, married, 60 years old on entrance August 31, 1911.

Right side hemiplegic. Became demented and childish; memory defect; disorientation. Died March 15, 1912.

B. *Representing second generation.* No. 19766. Son of A. 28 years old on entrance September 12, 1914.

Depressed, listless, wanders at night. Occasionally excited and boisterous. Later, became dull and apathetic. Grasp on surroundings poor. Hallucinations of hearing. Refuses to work. Catatonic. Gradually grew weaker. Discharged May 8, 1915, because of poor physical condition. Died shortly after, at home. Dementia praecox, hebephrenic.

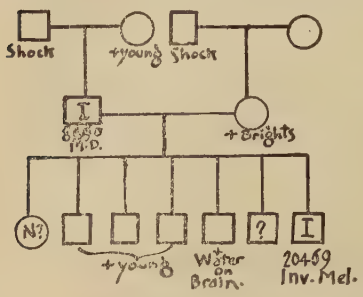
FAMILY GROUP NO. 70

A. *Representing first generation.* No. 8690. Male, married, 58 years old on entrance October 30, 1882.

Father was insane. Had a short attack of mental disease at 38, recovered.

This attack one year before. "Elastic in feeling, talkative, incoherent, excited, was destructive." Died of apoplectic stroke in 1882. In this case

the initial psychosis resembles manic-depressive though this cannot be definitely decided on the evidence. The organic brain disease seems to have been merely an incident which closed the life.



FAMILY NO. 70

B. *Representing second generation.* No. 20649. Son of A. Divorced, 58 years old on entrance December 11, 1912. Did poorly in school. Had somatic delusions. Believed that blood was dried up. Throat was choked. There was no circulation in the head. All the organs were in bad condition. Hallucinated, markedly apprehensive, ate very poorly, agitated. Died March 26, 1913. Diagnosis of involution melancholia.

Summary. It will be seen, both by the chart and by the above description of the case of the father, that the organic brain disease played no essential part and was merely incidental. It is probable that manic-depressive insanity is the proper diagnosis in the ancestor's case and involution melancholia in the descendant.

FAMILY GROUP NO. 71

A. *Representing first generation.* No. 6506. Male, married 51 years old on entrance April 5, 1876. Had a shock with right hemiplegia. Died April 28 of organic brain disease.

B. *Representing second generation.* No. 12409. Daughter of A. 32 on entrance March 12, 1895.

Onset at 31. Suicidal, sleepless, depressed, resistive. Said she had no insides. Food and drink could not nourish her. Nihilistic ideas marked. Improved; discharged May 28, 1898. Was well in the interim.

Re-entered July, 1909, with the same ideas. Incoherent, agitated, depressed, no dementia, no hallucinations. Was discharged August 20, 1909, about to die. Died at home in a few days.

Summary. A complete history reveals the fact that the wife of patient A (mother of B) came of a psychopathic family and that several of the family were either insane or nearly so. It is therefore possible that the disease of the daughter was probably related to the mental disease of the mother's family rather than to the organic disease of the father.

FAMILY GROUP NO. 72

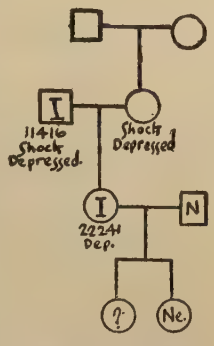
A. *Representing first generation.* No. 11416. Male, married, 61 years old on entrance April 14, 1892.

Had paralytic shock and later two others. Since then depressed, suicidal, and occasionally noisy. Stubborn, sensitive, bedridden. Had delusions that women came into the room and disturbed him. Was markedly confused. Had hallucinations of hearing and became completely demented towards the end. Died April 15, 1893.

Had one brother who died of cancer, one sister died of shock. *His wife died of cerebral hemorrhage, had been depressed before the attack. Her father committed suicide in a depression.*

B. *Representing second generation.* No. 22241. Daughter of A. 51 years old on entrance November 18, 1915.

At menopause former mild asthma became severe. Always of nervous, sensitive, and easily depressed disposition. Because of the asthma became addicted to drug habit. Broke this off by stay in the Westboro State Hospital. Became depressed, agitated, threatened suicide.



FAMILY NO. 72

In the hospital, no hallucinations, no dementia. Good insight into condition. Declared that her depression came because of the asthma, a feeling that she was incurable and would be a burden to everybody. Died February 15, 1915. Autopsy, by writer, showed some pulmonary condition.

Summary. It is distinctly more probable that the psychopathic condition of the daughter was related to the mental state of the mother and maternal grandfather than to the organic brain disease of the father.

FAMILY GROUP NO. 73

A. *Representing first generation.* No. 20012. Male, married, 76 years old on entrance May 14, 1912.

Patient had a cerebral hemorrhage at 28 years of age. Was always helpless, somewhat demented, and excitable since that time.

On entrance to the hospital, in 1912, had right hemiplegia. Memory disturbance for recent events, irritability and confusion. Was transferred to Howard, R. I., and died there. Diagnosis, organic brain disease.

An outstanding feature of this case is the very early onset of cerebral arterial disease.

B. *Representing second generation.* No. 21059. Daughter of A. 32 years old on entrance December 31, 1913.

Three distinct attacks of which this is the third, each marked by depression, confusion, retardation, and gradual improvement to normality with clear interim. She was sent to Howard State Hospital in January, 1914, and recovered later.

Group F

The following group comprise those cases in which alcoholism is held responsible for the psychosis in the ancestor.

FAMILY GROUP NO. 74

A. *Representing first generation.* No. 15425. Male, married, 65 years old on entrance May 3, 1902.

He had a first attack at the age of 40. This second attack was a pure case of delirium tremens with tremor, visual hallucinations, confusion and rapid recovery. Was discharged May 16, 1902.

B. *Representing second generation.* No. 18747. Son of A. 40 years old on entrance January 25, 1909.

Patient had a typical short acute alcoholic hallucinosis. He heard voices threatening, was coherent, had no marked clouding of consciousness, entertained delusions of persecution based on hallucinations. The recovery was complete and he was discharged August 7, 1909.

FAMILY GROUP NO. 75

A. *Representing first generation.* No. 20288. Female, married, 55 years old on entrance August 13, 1912.

Two sisters are said to be neurotic. The mother died of cancer. Paternal side negative.

Patient acquired syphilis shortly after marriage. Took drugs to relieve pain. Has been a drug habitue since, also alcoholic. Showed no mental symptoms except a slight confusion for a short period after entrance to the hospital. Showed decided signs of nervous syphilis in ptosis of the left eye-lid and changes in the reflexes as well as positive Wassermann in blood and spinal fluid. There were, however, no mental symptoms whatever after a short stay in the hospital, and the diagnosis of alcoholism and drug habit in a syphilitic seems without doubt.

B. *Representing second generation.* No. 20467. Daughter of A. 32 years old on entrance.

Had a double-sided inheritance. Her father was an alcoholic as well as the mother. The father's father committed suicide. She has one sister who was said to be insane.

Nevertheless, on entrance she presented merely the symptoms of intoxication with alcohol. Recovered quickly and was discharged within the period of observation, 10 days, as not insane—alcoholism.

Summary. Here we have an interesting fact that in addition to the alcoholism in the mother there was alcoholism on the part of the father and a decidedly neurotic trend through both families. Nevertheless, the daughter presented merely the symptoms of alcoholism.

FAMILY GROUP NO. 76

A. *Representing first generation.* No. 6103. Male, 54 years old on entrance June 30, 1875.

History of intemperance. A distinct and plain case of delirium tremens, from which he was discharged as completely recovered August 13, 1875. He died at 58 of apoplexy.

B. *Representing second generation.* No. 20100. Son of A. 55 years old on entrance April 58, 1912.

For 20 years had occasional insane attacks following alcoholism.

In the hospital showed the characteristic symptoms of a short acute alcoholic hallucinosis followed later by the surly temperament, irritability, general untrustworthiness, and mendacity of the chronic alcoholic. He died of pulmonary tuberculosis.

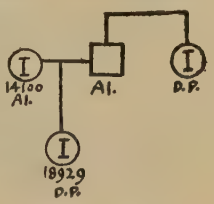
The above three cases are characterized by the fact that an alcoholic insanity in the ancestor was followed by an alcoholic insanity in the descendant. The next three show a somewhat different conclusion.

FAMILY GROUP NO. 77

A. *Representing first generation.* No. 14100. Female, married, 55 years old on entrance February 23, 1899.

Patient had an acute alcoholic hallucinosis of characteristic symptomatology, that is, hallucinations of hearing, delusions of persecution founded on the same coherence. No clouding of consciousness and gradual recovery.

B. *Representing second generation.* No. 18929. Daughter of A. Single, 34 years old on entrance October 18, 1909.



FAMILY No. 77

Onset at 30. Developed fantastic hallucinations and delusions of persecution of wide-spreading nature. Impressive. Well oriented. Said that the devil caused spirits to incite Catholics against her. Mentioned the Pope of Rome, Theodore Roosevelt, and certain local celebrities as being in a plot against her. The plot was to ruin her character, seduce her, cause her to steal and to eat dirt. From time to time changed her delusions with startling rapidity. Emotional tone rather good-natured, perhaps somewhat indifferent. Took a lively interest in her comfort, sometimes destructive. Reacted to auditory and visual hallucinations. Never demented. Sent to Medfield State Hospital where she is at present. Hospital diagnosis of dementia praecox, possibility of the disease belonging to the paraphrenia confabulans type.

A closer examination of the family history shows that the father was also an alcoholic and that his sister died at Danvers State Hospital with a diagnosis of chronic mania, which, in the days when the diagnosis was made, meant merely dementia praecox, as an examination of the records show. In other words, there is a paternal inheritance of mental disease as well as a maternal inheritance of alcoholism to be considered as related to the psychosis in this patient. It is more likely that the paternal inheritance determined the type of psychosis than that the maternal inheritance did.

FAMILY GROUP NO. 78

A. *Representing first generation.* No. 11955. Female, married, 40 years old on entrance September 26, 1893.

Father died insane. The mother was intemperate.

Patient always had a bad temper, seclusive, irritable. A heavy drinker. Recently had hallucinations of hearing with delusions of persecution and reference. Decidedly immoral.

In hospital, excited, delusions of persecution, hallucinations of hearing, from which she promptly recovered and was discharged April 7, 1894.

Re-entered at 43, October 13, 1896. Excited. Accuses the husband of incest with his daughter. Talks out of the window in reaction to hallucinations of hearing. Is coherent, well oriented.

In hospital recovered quickly and discharged. Acute alcoholic hallucinosis of psychopathic basis.

B. *Representing second generation.* No. 12083. Daughter of A. 19 years old on entrance May 1, 1896. Typical hebephrenic dementia praecox of the bench type. Demented, apathetic, indifferent. Occasionally reacts to hallucinations. Cyanosis of hands and feet. Was taken home for a short stay and re-entered, No. 17994.

In hospital at present, completely demented.

Summary. In this case we have an alcoholic patient with a psychopathic ancestry, of a peculiar, seclusive, paranoid temperament who, under the influence of alcohol develops acute alcoholic hallucinosis. Her daughter shows typical dementia praecox. It is more probable that the psychosis in the daughter is related to the psychopathic ancestry and the peculiar temperament of the mother than to the latter's alcoholic habits.

FAMILY GROUP NO. 79

A. *Representing first generation.* No. 19586. Female, married, 46 years old on entrance October 14, 1913.

On the birth of first child was out of head for a few days. Became a heavy drinker and shortly before entrance had hallucinations both of sight and

hearing, marked delusions directed against husband, and mild delusions of persecution. Recovered very quickly and shortly after admission to hospital appeared normal. Was discharged in a month.

Re-entered, No. 20981. At that time was sick bodily. Had a leaking valve of the heart and chronic nephritis. Hallucinations and delusions were marked. Memory was poor. Incoherent conversation; disoriented. Occasionally irritable but generally indifferent and apathetic. Died. Autopsy, held by writer, showed cardiorenal disease of severe grade.

In this case we have a patient who at three different periods of her life reacted to somatic influences by psychosis. In the first instance, after childbirth; in the second, after alcohol; in the third, while suffering from incompensation of the heart and renal disease. While the psychosis after alcohol was distinctly an acute alcoholic hallucinosis, we have to deal with a susceptibility to toxic influences of an unusual kind.

B. *Representing second generation.* No. 22259. Son of A. Single, 22 years old on entrance November 23, 1915. In hospital at present.

Has been alcoholic. In hospital, apathetic, delusions of persecution of a marked yet incoherent type. Active hallucinations of sight and hearing. Religious ideas of a grandiose type.

Later, became very negativistic, mute, sullen and demented. At present sits around with head bowed, saliva dripping from mouth, takes no interest in environment, has to be led to and from his meals. Diagnosis, hebephrenic dementia praecox.

Summary. It is probable that the dementia praecox in the latter patient's case is not related to the alcoholism of the mother but to the peculiar bodily construction which made her react to toxic influences by a psychosis.

Group G

The following group of cases in which the psychosis of the ancestor which occurred at the senium and was classified as senile is of decided importance and interest. It will be shown that diverse types of disease followed in the descendants. The contention is that this is largely because the term "senile psychosis" is loosely used to include a group of diseases which are found at any age, but which receive a special coloring from the psychology of the senium:

FAMILY GROUP NO. 80

A. *Representing first generation.* No. 16554. Female, widow, 84 years old on entrance September 8, 1904.

Always very jealous, odd, suspicious, sensitive. Around 80 developed delusions of reference and persecution; said a dog has been trained to bark

to annoy her. The neighbors wished to get her out of the neighborhood. Believed the food had been poisoned. Became destructive, excited, and sleepless. No hallucinations.

In hospital, the persecutory ideas spread out against other people in the hospital. Memory defect was very slight. Died one year later, Diagnosis of hospital, senile dementia.

This diagnosis seems to be erroneous. The entire trend of the personality was paranoid. The personality throughout life had been paranoid. At the latter end of her life a paranoid psychosis developed. I venture the diagnosis of a senile paranoid condition.

B. *Representing second generation.* No 12089. Son of A. 34 years old on entrance March 2, 1894, 29 at onset.

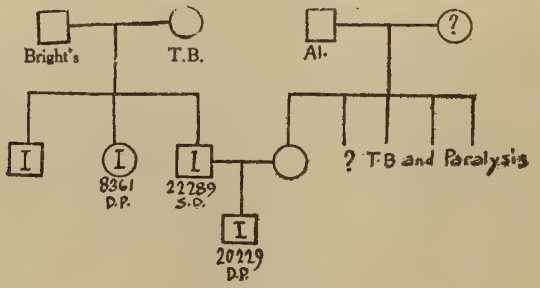
One of five children, four of whom died in infancy.

Patient presented typical dementia praecox with sexual paranoid ideas, that is to say, he was impotent through the machinations of others. He had hallucinations of hearing and of smell. Impulsive and destructive acts in response to his hallucinations. Became apathetic and indifferent. Moderately demented. Transferred May 1, 1896 to Medfield State Hospital. Diagnosis, paranoid dementia praecox.

Summary. A paranoid psychosis in the ancestor developing late in life followed by paranoid dementia praecox in the descendant, developing earlier.

FAMILY GROUP NO. 81

A. *Representing first generation.* No. 22289. Male, 73 years old on entrance December 18, 1915. Notes exceedingly scanty because patient died within week. Apparently an old senile dementia with destructiveness, excitement, confusion, marked memory defect, and great bodily weakness.



FAMILY NO. 81

B. *Representing first generation.* Nos. 8361 and 10556. Sister of A. First admission was at the age of 33, July 13, 1881. Was discharged April 1, 1882. Second admission at the age of 42, May 9, 1889. In hospital at the present time. Noisy, destructive, markedly hallucinated, and deluded, very quickly dementing.

At the present time presents the typical picture of a demented praecox patient. Diagnosis, dementia praecox.

C. *Representing second generation.* No. 20029. Son of A, nephew of B. Married, aged 30. In hospital at present time. A very demented, seclusive patient with paranoid ideas and hallucinations of hearing and sight. Negativistic, absolutely apathetic and indifferent.

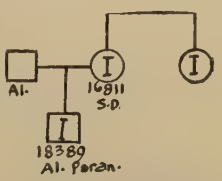
A further examination of the family history in this case shows the following: The paternal side, as above presented (see chart), showed senile dementia in the father, dementia preacox in the sister, and we know, in addition, that the father's brother was "insane" and his uncle as well. The paternal grandfather died of Bright's disease, the paternal grandmother of tuberculosis. The maternal side, which did not appear in the hospital, shows alcoholism in the maternal grandfather and ill-defined, ill-described nervousness in the mother.

FAMILY GROUP NO. 82

A. *Representing first generation.* No. 16811. Female, married, 62 years old on entrance March 25, 1905.

Father died early. Mother died of old age. A maternal cousin is feebleminded. One sister, said to be like the patient.

At 57 patient began to be forgetful, showed nocturnal restlessness, hallucinations of hearing, untidy, became demented and irritable. The note, "No memory" in the records tersely describes her mental condition. She died October 14, 1906, of dysentery. Diagnosis, senile dementia.



FAMILY NO. 82

B. *Representing second generation.* No. 18389. Son A. Married, 58 years old on entrance September 30, 1908.

Father died of old age. The entire paternal side shows marked alcoholism. 20 years before, at 38, patient had delirium tremens. Always nervous, had blue spells, and drank heavier lately. Had delusions of jealousy against wife. No hallucinations. Recovered promptly, worked steadily, and was discharged November 19, 1909, recovered.

Summary. The ancestor in the hospital had senile dementia. Her son, following a depression, became alcoholic, developed a paranoia and recovered. It is, however, shown that the father's side was exceedingly alcoholic and it is more probable that the alcoholism in the son with a consequent transitory psychosis was more definitely related to this than to the senile dementia of the mother.

FAMILY GROUP NO. 83

A. *Representing first generation.* No. 13647. Female, 71 years old on entrance December 6, 1897.

Onset said to be at 67. For years noisy, difficult to manage, very aggressive.

In hospital, markedly incoherent, irrelevant, destructive, disoriented, and unmanageable. Sings and talks to herself, finally became decidedly apathetic. Died at 73.

On the face of it, psychosis is senile dementia, but certain trends in the character for years and the markedly aggressive and finally apathetic character of the psychosis point towards other possibilities.

B. *Representing second generation.* No. 19000. Daughter of A. Married, 40 years old on entrance December 1, 1909. In hospital at present time.

FAMILY NO. 83

As throwing light on A's character, it is stated that her husband, the father of this patient, deserted the family early. One sister died of tuberculosis. Two sisters melancholy, but not in hospital.

This patient was married three times, the first two husbands dying. There are three children. The change occurred after the first pregnancy. Onset really at 27. Had several attacks during which she was confined to various hospitals. These attacks were usually marked by very great excitement, delusions of grandeur, but refusal of food; distinctly aggressive conduct with a gradual betterment to the point at first of recovery, and later only to the point of moderate remission. Following the first attack there was an intermission of two or three years. The diagnosis of three hospitals, including the McLean Hospital at Waverly, Butler Hospital of Providence, and the Taunton State Hospital, at first was manic-depressive insanity. The last attack, occurring at the age of 40, was marked by delusions of reference and persecution, decided hallucinations of sight and hearing, and a profound and rapidly progressive dementia, so that at the present time there is no question as to the diagnosis of dementia praecox. She is catatonic, resistive, seclusive, and very markedly hallucinated.

Summary. In this family we have a psychosis occurring at the senium with some characters not unlike the psychosis in the daughter, which is decidedly that of dementia praecox. There is in the mother's case, destructiveness, aggressiveness, wild excitement, and finally

apathy. In the daughter's case there are periodic attacks which at first diagnosed as manic were more like catatonic and later a distinct dementia praecox. Though the psychoses differed in many respects, in the respect of hostility to the environment and marked excitement they are alike, and in the respect that both terminated finally in apathy and dementia they are also alike. In the first individual, the psychosis occurred later and ran a rapid course; in the second individual, the psychosis started early and ran a protracted course.

FAMILY GROUP NO. 84

A. *Representing first generation.* No. 13711. Male, married, 82 years old on entrance March 2, 1888.

Onset at 77. Aggressive, demented, irritable, decidedly grandiose, and boastful. Was quarrelsome, talkative, restless. Was completely demented toward the latter part of stay. Died of cystitis in 1901 at the age of 95.

B. *Representing second generation.* No. 17484. Son of A. Widower, 56 years old on entrance September 27, 1906.

Onset was sudden. Was very exalted, boastful; said he was a messenger of God with instructions to clean up the plague spots. (Moral plague spots.) Sings and talks in Salvation Army tone. Has ideas of poisoning and refused food for a week. By December 22, 1906, no such ideas could be elicited from him and he was discharged.

Since then he has written queer letters to his people and to the authorities in which it is very evident that the grandiose ideas of boastfulness, the feeling of exaltation and power still persist.

Summary. It is difficult to classify either case in the Kraepelinian terms. In the one case, at the senium, patient became grandiose, boastful, irritable and finally demented. In the second inidvidual, we might call the psychosis senile dementia, but that would be disregarding the difference between this and other cases of senile dementia. The son, at 50, suddenly became exalted, boastful, and talkative, from the acute symptoms of which he recovered, but enough was left to show that a substratum of the psychosis still persisted. The diagnosis of manic-depressive insanity was made in in the latter case. This diagnosis seems doubtful to me in that no true recovery followed, that the grandiose and boastful characters persisted, and that while he was talkative there was no flight of ideas. A paranoid condition of some kind is to be considered. In certain essential features, the psychosis in the one individual strongly resembled the psychosis in the second.

FAMILY GROUP NO. 85

A. *Representing first generation.* No. 5370. Male, married, 63 years old on entrance September 24, 1874.

"Mild with occasional violent symptoms. Has noises in the head. People are talking to him."

In the hospital excited and destructive.

Later, very much depressed, worried, self-distrustful. No hallucinations. Was discharged, improved, March 15, 1875.

Re-entered April 2, 1875, No. 5954. Very deluded, suspicious; believes people are against him and wish to do him harm. Eats poorly, is agitated. Discharged September 1, 1875, unimproved. Psychosis unclassified, possibility of involution type of mental disease.

B. *Representing second generation.* No. 21470. Son of A. Married, 76 years old on entrance June 27, 1914.

Has paralysis agitans with occasional periodic attack; rambling in talk; filthy; speech entirely undistinguishable so that psychotic type cannot be determined. Evidently, however, demented. Died at 77.

Summary. In this family group the reverse of anticipation occurred. In the first patient, an involutional type of psychosis is followed by paralysis agitans with probable senile dementia in the descendant.

FAMILY GROUP NO. 86

A. *Representing first generation.* No. 19922. Female, widow, 74 years old on entrance December 15, 1911. In hospital at present.

Onset at 70. Patient a heavy drinker. Has hallucinations of sight and hearing. Shows distinct dementia, restlessness, rather suspicious attitude, poor judgment, and untidiness. Diagnosis, senile dementia on alcoholic basis, question.

B. *Representing second generation.* No. 22143. Son of A. Married, 37 years old on entrance March 19, 1914. Was a drinker. Had pneumonia just before hospital entrance. Developed visual and auditory hallucinations, marked confusion and restlessness, delusions of infidelity. Irritable emotional tone. Recovered shortly after the temperature had dropped to normal; discharged. Diagnosis of acute alcoholic hallucinosis with a question of febrile psychosis; in other words, a toxic insanity is the diagnosis.

C. *Representing second generation.* No. 17697. Son of A. 25 years old on first admission when he showed hallucinations, delusions of persecution; marked apathy; sits around all day, doing nothing, with head bowed on hands and apathetic. Considered feeble-minded for many years. Is in the hospital at the present time. Diagnosis, dementia praecox, hebephrenic type.

D. *Representing second generation.* No. 18378. Daughter of A. Married, 36 years old on entrance August 4, 1908. Patient showed a decided general paresis with typical physical and mental signs. History of syphilis.

Summary. A senile or possible chronic alcoholic hallucinatory dementia in the ancestor. One son acute alcoholic hallucinosis, another dementia praecox hebephrenic, and a daughter, who had perhaps better be excluded from consideration, showed general paresis. Anticipation is marked in these cases.

FAMILY GROUP NO. 87

A. *Representing first generation.* No. 20014. Married, male, 82 years old on entrance January, 1913. His mother was demented in old age. A sister insane for a short time.

Patient was married three times. All of children died young excepting the son, B. Patient was an engineer and when old became a janitor. Always an efficient man, but seclusive in temperament. Onset at 75. Was melancholy. Attempted suicide.

Later, became irritable, abusive, indecent. Developed delusions of persecution and delusions of grandeur. Later, became demented and occasionally showed marked negativistic traits. Further hospital notes state "memory poor, vague hallucinations, delusions of persecution, hallucinations of sight and hearing." He died March 17, 1913, completely demented.

B. *Representing second generation.* No. 10612. Son of A. Single, 27 years old on entrance August 5, 1889. Very noisy, violent, suicidal. Cut his throat. Onset with a loss of appetite and sleeplessness.

In hospital, depressed, excited, fed by stomach tube on account of neck wound. No hallucinations. Recovered December 11, 1889; discharged. Diagnosis, probable manic-depressive insanity, depressed phase, possibly of catatonic attack.

Summary. In the case of the father, psychosis passed through definite stages, from depression, delusions and hallucinations to apathy and dementia. If the psychosis had occurred at 30, no one would question dementia praecox as the diagnosis.

FAMILY GROUP NO. 88

A. *Representing first generation.* No. 10035. Male, married, 68 years old on entrance March 27, 1887. Onset at 66. Violent, at times suicidal, tried to poison himself, very sleepless, restless, and destructive.

In the hospital, hallucinations of hearing, with marked delusions of persecution directed against his wife and sons. Very much agitated, not particularly depressed. Resistive. Died of pneumonia May 16, 1887.

A psychosis of paranoid character in that the most marked features are hostility towards wife and sons with violence and attempt at suicide. Suicide in such cases may be interpreted as a means of ending an intolerable situation of the persecutory type.

B. *Representing second generation.* No. 20885. Daughter of A. Female, single, 54 years old on entrance August 19, 1913. Had epilepsy for 15 years. For some time past has been restless and agitated.

In hospital, showed psychomotor activity, destructiveness, excitability, and resistiveness, hallucinations of sight and hearing, delusions of persecution and apprehensiveness. A few epileptic attacks in hospital with a psychosis apparently not depending upon them. Diagnosis of paranoid dementia praecox.

Summary. In ancestor, a paranoid psychosis occurring at the senium. In descendant, a paranoid psychosis occurring near the menopause. In the first, to be diagnosed as senile paranoid condition; in the second, as paranoid dementia praecox.

FAMILY GROUP NO. 89

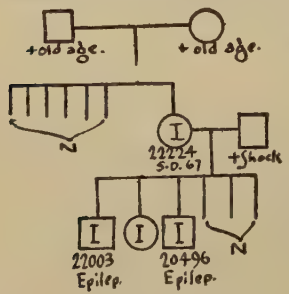
A. *Representing first generation.* No. 10348. Male, 84 years old on entrance May 17, 1888. Onset at 80. Utterly demented, incoherent, filthy, restless and weak. Died June 4, 1888. Diagnosis, senile dementia.

B. *Representing second generation.* No. 18077. Son of A. Married, 77 years old December 10, 1907. Showed practically the same symptoms as father. Died January 16, 1908.

Summary. Senile dementia is the diagnosis in both cases. The family history states indefinitely that there was mental disease in the collateral relatives.

FAMILY GROUP NO. 90

A. *Representing first generation.* No. 22224. Female, 63 years old on entrance November 6, 1915. Typical simple senile with childishness, untidiness, irrelevancy, dementia, and occasional destructiveness. Hallucinations and delusions not present. Died January 24, 1916, of heart disease.



FAMILY NO. 90

B. *Representing second generation.* No. 20496. Son of A. Single, 40 years old on entrance January 7, 1913. Epilepsy since childhood. Following last attack became hallucinated and deluded. Had religious delusions, and died October 27, 1913, of pneumonia, following an attack. Diagnosis, epileptic insanity.

C. *Representing second generation.* No. 22023. Son of A. Single, 21 years old on entrance June 11, 1915. Feeble-minded from birth, epileptic from birth. Died in attack. Autopsied by present writer. Showed cerebral congestion,

hypostatic condition in lungs and exceedingly small body and organs, including liver, kidneys and spleen. Brain of average size.

Summary. Senile dementia in the ancestor, epilepsy in two descendants.

FAMILY GROUP NO. 91

A. *Representing first generation.* No. 8692. Male, 75 years old on entrance August 22, 1882. Fell down flight of stairs and since then has been insane. Confused, incoherent, marked memory defect. Died April 27, 1884. Throughout stay was completely demented and considered a typical case of senile dementia.

B. *Representing second generation.* No. 19310. Son of A. 63 years old on entrance July 27, 1910. Disease probably of many years' duration. Was a pauper. Believed people were blowing things on him through the radiators of the almshouse. Threatened to kill his persecutors. Threatened suicide if persecution was kept up. Well oriented as to time, place and person; memory intact. Hostile and suspicious, emotional tone. Occasional hallucinations of hearing. October 14, 1910, discharged to the Foxboro State Hospital.

Summary. In the parent, senile dementia; son, paranoid condition, probably dementia praecox.

FAMILY GROUP NO. 92[4]

A. *Representing first generation.* No. 19741. Male, 69 years old on entrance December 1, 1910. Onset at 68. Very deaf, poor vision. Disoriented, memory markedly impaired, very noisy at times, untidy, and restless. In hospital at present. Considered typical senile dementia.

B. *Representing second generation.* No. 19345. Daughter of A. 42 years old on entrance August 24, 1910. Onset many years before. Has marked delusions of persecution, aggressive, disoriented, excitable, hallucinations of sight and hearing. Grew demented. Sent to Medfield State Hospital September 6, 1911. Paranoid dementia praecox.

I

The following 2 cases are reported in detail because of the detailed study that it has been possible to make.

In the first family there are three generations involved, all of whom are at the present time in the hospital. In the second, two generations are involved, both of whom are at present in the hospital and moreover, a very complete family history of the non-insane members of the family has been made.

[4]Cases 93, 94, 95, are omitted as obscure and non-psychiatric.

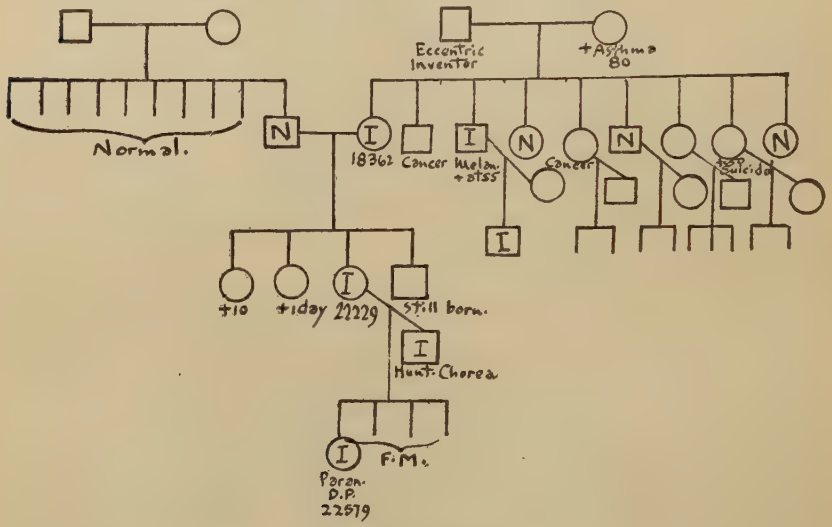
FAMILY GROUP NO. 96

A. *Representing first generation.* No. 18362. Female, married, 55 years old on entrance July 27, 1908. In hospital at present.

Father of patient was eccentric all of life; thought to be queer and somewhat unbalanced. Spent a good deal of his spare time on secret inventions which never materialized. Though not an educated man had a mania for astronomy.

The mother's side is said to have been entirely normal.

The patient was the third in a family of eight children, most of whom died early. One sister committed suicide. One living sister was quite nervous. She married at the age of 21. Husband said to be normal. Pregnancies were four, three of which were abortions and one living child, B.



FAMILY NO. 96

Always said to be of exacting and scrupulous nature. At the menopause the first mental symptoms appeared. Had indistinct hallucinations of hearing at that time. At 51 she had crying spells and was depressed. Felt incapacitated for work. Attempted suicide. "Recently hallucinations have become very active." She believed that her husband had committed some crime and attempted to give the police officers of the town some money for it. Believed her husband was unfaithful to her and asked for divorce proceedings. Finally, just before entrance, mistook her husband (as the latter slept in the same bed) for another man and crying out against him, struck him with a rolling pin.

Physical examination negative: Wassermann examination negative.

Summary of mental examination. Marked auditory hallucinations to which she reacts, but whose character she will not admit. At one time, however, she threw her wedding ring out of the window because the voices advised her to do so. Suspicious. Answers coherently, but with some preliminary hesitation as if deciding whether it were wise to answer. Delusions of persecution on the part of husband are marked as well as delusions of infidelity. Memory intact. Orientation not impaired. Quiet; tractable. On October 23, 1909, she was discharged for a trial of 180 days against advice.

Was returned February 23, 1910. Delusions of persecution against husband and woman up-stairs more fixed and more specific. Accuses her husband of definite infidelity and definite attempts to murder her. Says that a general in the United States Army knows all about her and her husband. Says that she has fined the Roman Catholic Church and that the fine has got to stay. Abusive to physicians; extremely suspicious and irritable. Says that she is the station for the wireless telegraphy of the Marconi system and when she receives a message it goes to the scientists and this is carried all over the country. She receives messages from all over the world. Hallucinations of hearing are active.

In 1910, no dementia noted. In September, 1910, the note states "that the food and milk in the hospital contained poison; that the superintendent has been changed into a Mr. Wood." Extremely surly and insulting. Believes that the Catholic Church is persecuting her. Hallucinations of hearing.

From that time to the present her condition has been progressive. She takes little interest in her environment, but when approached becomes violent, extremely vile and insulting, and decidedly antagonistic and suspicious. Entertains delusions of persecution which, however, are vaguely expressed. Reacts to auditory hallucinations. Is believed to be demented, though this cannot be ascertained because of her extreme antagonism. On entrance, involution psychosis was the diagnosis; at the present time, paranoid dementia praecox.

B. *Representing second generation.* No. 22229. Daughter of A. Married, 38 years old on entrance November 8, 1915.

Married a man who is now in the hospital with Huntington's chorea and who comes of a Huntington's chorea family.

Early history. Sickly in early life, having had practically all the so-called children's diseases. She was not markedly backward in school, attending high school. Had five children, of whom one is at present a patient in this hospital, two are at present in the Wrentham State School for Feeble-Minded, the fourth died at the age of thirteen months, and the fifth, at present six years old, looks feeble-minded. Has had three miscarriages. Separated from her husband five years ago because of his physical trouble.

Physical examination of patient is negative.

Summary of mental examination. Believes that a gang of Jews are persecuting her because one of them, attempting to make love to her, was repulsed. She has felt obliged to purchase goods of a Jewish merchant in the town because she felt that the Jews would kill her unless she did so. Has delusions of poisoning. Thinks that God gave her signs through a ring

which was on her hand as to what she should do. Heard voices moaning and groaning outside the window and saying, "Tell, tell, tell." Believes that her whole family will be killed by the Jews, one of whom, she says, made a mark of the cross on her neck, and she points out a small red mark on her neck as the center of it. Very poor retention of school knowledge. Conversation is relevant, fairly coherent. She is quiet, tractable, and seclusive.

Somewhat later, in hospital, said that she saw the devil on the ceiling. The following is a sample of her conversation: "I think the end of the world is coming because the German girl came to me, I believe, on account of this Jew. I am the third child. My father, my mother, and my husband also were. It is also God the Father, God the Son, and God the Holy Ghost—three in one. My husband is the cause of it all. I don't know whether it is heaven or hell. The Catholics they do badly." Questioned: "How is your husband the cause of it?" "Because he brought the Jew into me." Emphasizes the work "into."

Later on, it became difficult to get a direct answer to questions as she wandered markedly in her conversation and grew demented. "She sits around on the ward, does but very little, will not work. Smiles in a silly manner. Hallucinations and delusions quite prominent. Tractable, shows no effect when talking of her delusions."

C. No. 21683. Husband of B. At present in hospital. Is a typical case of Huntington's chorea.

D. *Representing third generation.* No. 22579. Daughter of B. and C. Single, 19 years old on entrance June 26, 1916. Always somewhat mentally deficient. Very difficult for her to do her school work. Obtained a position in a factory. While visiting her mother at the hospital expressed so many peculiar ideas that it was deemed advisable to have her committed to this institution.

Physical examination is negative. No signs of Huntington's chorea. No tremor of any kind. In fact, physically is an attractive looking girl.

Very marked delusions of persecution and reference of an incoherent, absurd type. Says that the wheels of the electric cars are able to read her thoughts and grind them aloud so that everybody on the street knows about them. Imagined in the Pearl Works, where she was employed, that the machines did the same thing and her fellow employees knew just what was in her mind and made great fun of her. She thinks a man who is married and some 30 odd years old has been in love with her, but lately has jilted her for an older woman. Memory intact; perfect orientation; grasp on surroundings good. Emotional tone is silly; childish and complacent, for she accepts the commitment to the hospital as a perfectly natural and proper thing. Intellectually measures about eight years. Her answers are relevant though somewhat incoherent. No insight. Diagnosis of paranoid dementia praecox on a feeble-minded basis made.

Summary. A glance at the chart shows the following: The husband of the first patient comes of a stock apparently normal. The first patient, A, had a father who was considered eccentric and

queer. Mother died of asthma. In her fraternity was a brother who was insane at about 45. He married a normal woman and has an insane son. Two of her sisters died of cancer. One committed suicide at 50, was depressed. Three other members of the family are said to have been normal, though little is stated regarding them. In the second generation, issuing from this pair, there were three miscarriages, and an insane daughter. This insane daughter married a man with Huntington's chorea, at present in the hospital, and from them there issued four living children, three of whom are definitely feeble-minded and one is at present in the hospital. The three females in this hospital, representing a first, second, and third generation, have identical psychoses—paranoid delusions, hallucinations of sight and hearing, and dementia characterize them—and a diagnosis of paranoid dementia praecox can be regarded as very definitely established in their case. The psychosis appeared earlier in each generation, and it does not seem probable that in the case of the young woman representing the third generation the fact that her father had Huntington's chorea influenced her mental state and the time of its appearance in the least.

FAMILY GROUP NO. 97

A. *Representing first generation.* No. 10217. Male, married, 34 years old on entrance November 12, 1887. Onset of psychosis two months before when he attempted to shoot himself under the belief that he must make a sacrifice. "Mind dwells constantly upon religious topics. He is troubled lest he is not a Christian. Everything about him is changeable and uncertain. His Heavenly Father tells him to do certain things—to move, to act, to pray. He hears persons about him making uncomplimentary remarks. Is jealous of his wife and suspicious of people in general."

Gradually improved, delusions and hallucinations disappeared. Was easily elated and depressed upon discharge, June 26, 1888.

History obtained at the time of first commitment stated that he had always been nervous. Had been considered peculiar since he was a boy. Has taken liquor for many years to stimulate him up to his work.

Second admission. No. 13570. 39 years old on entrance September 26, 1892. Used alcohol in excess. "Did not fully recover from last attack, had been peculiar." He was depressed, confused, and emotional. *Masturbated openly.* Believed that many people were enemies to him and overheard them talking against him. He was quick to apply chance remarks not directed at him, to him. Worked fairly well. Was discharged to the Overseers of the Poor July 22, 1893, somewhat improved.

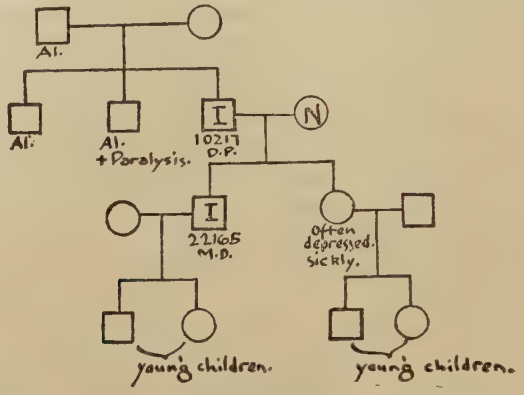
In the almshouse, his mental condition again became troublesome and he was sent to Medfield State Hospital.

In Medfield, showed increasing dementia, hallucinations of sight and hearing, delusions of persecution, and died after four years in hospital. Diagnosis, dementia praecox.

Despite the depression-like onset, the case seems to me clearly to be one of dementia praecox, and this is favored by first of all the temperament, which from the start was suspicious and peculiar, and by the termination of the disease in dementia with paranoid ideas and hallucinations before the age of 40.

B. *Representing second generation.* No. 14548. Son of A. Single, 22 years old on entrance October 18, 1900.

Mother bright by all accounts, and from personal interview a very normal appearing woman.



FAMILY NO. 97

Onset of this psychosis sudden. He was excited, talked about the great inventions he had made; said he had immense wealth, had a great work to perform for mankind. This followed an attack of grip.

In hospital, memory for past and recent events intact. Answers are interlarded with much irrelevance and no obvious association of ideas; for example: (What day of the month is this?) "A week ago Sunday was the 8th—today is the 16th. You can order me—I will do what you ask. I can get out of this room if I want to but I don't. I was low down, as low down as can be—I'm here to receive the test. You can't get any here to do the will of God. Whatever he may desire or she may desire—it may be a she-God. It is easier to walk forward from backward. The socks I have on are not necessary—the shirt and trousers are necessary. I received my inspiration last Thursday. I'm here to do the will of God. I'm at peace with God. When the time comes I shall have the power to build people up. The connecting link in their brains has been found." The following design is one which he wrote and which is explained as follows:

"We'll begin at the bottom—G-o-u-l-d. I feel it in my bones—I marked it with an eyelet taken from my drawers. Gould is the foundation. The

next is good—eagerness to give money away. The last is God—the heir of every man or woman whether he be male or female. When I started on the street railroad I wanted to work with an heir, to improve at every point. I wanted to be a division master and then a general manager. I didn't stop at general manager and then the idea of the money-changing machine came to me—money is now no object. Good-bye, God bless you—God bless everybody."

Throughout his stay he was very euphoric, happy, busying himself with everybody and everything on the ward, and continually talking. He wrote many letters, of which the following is a sample:

"To The Doctors: I am now ready to give you proof for any question you ask, and I would like to ask you a few.

"1st. What day of the month is it?

"2nd. Prove it.

"3rd. Can you prove that today is not Saturday, June 30? To simplify matters, can you prove that you have not slept through Wednesday, Thursday, and Friday? While you have been asleep you may have died a hundred times, and been resurrected each time. I dislike to have to make out that Christ, the 2nd, has been on earth in the disguise of 'H. S.', but until you can prove that I am not the second Christ, I think you have not the right to dispute it. Please compare my actions and words with those of Bible times. You may remark, 'But you use profanity.' My so-called profanity is nothing but prayers. I came here for proofs of certain statements which 'I' have made. That simple 'change-making' machine (the human system) can control the whole world by destroying the money as soon as received. *Prove* my statements incorrect if possible. To simplify matters we find that, referring to the Bible, '1000 years is but yesterday when it is past.' So by that today is June 27, 2900. Can you *prove* that 'H. A. S.' did not even write the Bible, under an assumed name? If so, how? I am not entirely satisfied with this place, so I would like my discharge as soon as possible. My time is valuable, as I said when I came here. How much have you wasted for me? None, but if I am kept here much longer you will compel *me* to waste some. As soon as you can prove any of my statements incorrect by a 'competent' judge, I will prove them correct, until then it is unnecessary. Do I go home today or not? Who is Christ, 2nd? With best Easter wishes.

"My word is as good as my note, and my note must be good for everyone has it. H. S."

Recovery took place at the end of six months and he was discharged October 13, 1900, recovered.

He was well in the interim between that discharge and September, 1915. At that time, following a period of intense heat, he again became much excited and entered the hospital. He had married in the interim of 15 years, had conducted his duties as a mail carrier, and performed his obligations as husband and father in what appeared to be a perfectly normal manner.

On entrance he was very active, talking loudly and swearing. A formal mental examination was impossible because of his entirely disconnected and irrelevant conversation. He was oriented as to time and place. Then went

on to say, "Station eight, station one." When "State Hospital" is suggested he says, "Oh, that's the catch." His spontaneous conversation is something like this: "Correct is right, Oh, I see the point—'pon my word. You don't see the point. You can't catch me on a one ninety-seven. Ah, ha! Oh, ho! Correct is right, correct is right. Talisman comes pretty near—pretty nice, pretty nice. You've got me. Correct is right." He became exceedingly noisy and excited and was kept in a continuous bath for a long period of time. During this time conversation could hardly be distinguished, it was so incoherent and rapid. He kept his hands in continuous motion. A sample of his conversation a month later follows: "Over the loop—perpetual motion, could you fellows start on. My own big toe—I'm dead and I'm dead, been dead through deadness—boy born in the other room, I heard one out there, I know—how are you in here? He's a fellow who opens the gate—two, three, four—one, two, three four; getting close out—three, four, five, six—seven, eight, nine, ten—eleven, twelve, thirteen, fourteen, fifteen—seventeen, twenty. Charge up anything you want to—not now—everything out. Wait till he gets out of the room—my nose and your nose like mayflowers. You measure the first class as this; correcting answered; one, two, three—four, five, six—seven, eight, nine—seventeen—twenty-three—thirty-five—forty-three—fifty-seven; probably out tonight—one, two, three, five, seven, sixteen—one, three, five, seven—four, three, two, one—one o'clock; open game of checkers and play it." (Men on ward playing checkers.) "All in comes, no-out goes. Freddie Foy out there waiting—Freddie is a fellow; yes, indeed; call anywhere."

During the height of the attack patient accepted every suggestion given to him as to the starting point of a new line of associations and presents as cardinal symptoms of manic-depressive insanity, manic-phase, psychomotor excitement, euphoria, flight of ideas with distractibility, and shallow current of thought; the associations being either suggested by sound or by some superficial remark. There were no delusions or hallucinations.

At the present time he is much improved, is becoming quieter, and conversation more relevant.

Summary. The case is quoted at some length because it seems like a typical manic-depressive case following dementia praecox, a transition which is not common. The chart of the family tree shows that patient has a sister who has short attacks of depression and is generally considered an extremely nervous woman, though by no means insane from the commitment standpoint. The father of the first patient was alcoholic, but no other definite ancestral abnormality has been found.

CHAPTER X

Conclusions in Relation to Vertitcal Transmission of Specific Mental Diseases

Problem, given a certain type of mental disease in an ancestor, what form of mental disease is to be expected in his direct insane descendant? It is understood, of course, that many of his descendants will not come to the hospital and what follows does not apply to them.

A. THE CASE REGARDING PARANOID PSYCHOSES

Of the two generation families in which the parent presented this condition, 3 (Nos. 25, 26 and 27, belong more properly perhaps to dementia praecox. I confess that I am unable in many *living* patients to differentiate between the varieties of paranoid condition. For this study I have taken but simple criteria. *Paranoia* is a psychosis with systematized delusions of persecution, fleeting hallucinations, no dementia. *Paranoid condition* may have less systematized delusions, but they relate definitely to a possible environment, hallucinations, though scanty, may exist, but dementia is not present. *Paranoid* dementia praecox is characterized by less systematized, generally incoherent, often absurd delusions of persecution. Hallucinations are marked and the condition tends toward dementia. This is, as I realize, a very crude analysis as compared with elaborate schemata used by some writers. I am sure that it is fully as accurate in practice. Of the 7 two generation families thus listed, the following is a summary.

No. 1. One of second generation had paranoid condition; her daughter in third generation, paranoid dementia praecox; fourth generation, catatonic dementia praecox.

No. 3. Generation one, paranoid condition of senium; generation two, paranoid condition of involution; generation three, paranoid condition of early adult life.

No. 4. Generation one, paranoid condition of senium; generation two, senile manic (?); generation three, indirect line, paranoid condition.

No. 23. Generation one, paranoid condition (dementia praecox); generation two, paranoid condition less marked.

No. 24. Three generation family: Generation one, senile paranoid condition; generation two, dementia praecox, catatonic, later paranoid; generation three, dementia praecox.

No. 25. Generation one, paranoid condition; generation two, paranoid.

No. 26. Generation one, paranoid dementia praecox; generation two, paranoid dementia praecox with a brother who is epileptic.

No. 27. Generation one, paranoia condition; generation two, paranoid dementia praecox one case; catatonic dementia praecox two cases.

No. 28. Generation one, paranoia; generation two, toxic psychosis.

No. 29. Generation one, paranoid condition (dementia praecox) with manic-depressive in some of fraternity, in others dementia praecox; generation two, paranoid condition (dementia praecox).

It will thus be seen that the following paranoid disease in the immediate ancestors, dementia praecox or a paranoid condition in the descendants has followed. The disease in the descendant usually commenced earlier and was generally worse than in the ancestor. *The paranoid psychosis itself in those cases where we have good histories* seems directly and distinctly related to character defect or peculiarities in person of the paranoid type, meaning by this term a personality characterized by suspicion, egotism, and either hostility to the environment (if the individual is strong) or seclusiveness (if the individual is weak).

If now these findings are compared with those of other workers we may formulate their results as follows: Paranoid condition in the ancestors breeds dementia praecox in the descendants. (Jolly (100), Luther (125), Krueger (116), Rosanoff (160), Albrecht (2)). The cases of all these writers, emphasize this conclusion in unmistakable and practically unanimous manner.

THE CASE REGARDING DEMENTIA PRAECOX IN THE ANCESTOR

No. 30. Generation one, dementia praecox; generation two, dementia praecox and epilepsy.

No. 31. Generation one, dementia praecox; generation two, epilepsy in psychopathic person with paranoid trend.

No. 32. Generation one, paranoid dementia praecox; generation two, paranoid dementia praecox.

No. 33. Generation one, dementia praecox; generation two, dementia praecox.

No. 34. Generation one, dementia praecox, in fraternity dementia praecox; generation two, dementia praecox.

No. 35. Generation one, dementia praecox; generation two, dementia praecox.

No. 36. Generation one, dementia praecox; generation two, normal imbecile.

No. 37. Generation one, dementia praecox with three of fraternity showing dementia praecox; generation two, moral imbecile.

No. 38. Generation one, dementia praecox; generation two, dementia praecox.

No. 39. Generation one, dementia praecox, one in fraternity showing dementia praecox; generation two, dementia praecox.

No. 40. Generation one, paranoid dementia praecox; generation two, three cases of dementia praecox.

No. 41. Generation one, dementia praecox in attacks, later chronic; generation two, dementia praecox in attacks, later chronic.

No. 42. Generation one, dementia praecox; generation two dementia praecox and feeble-minded.

No. 43. Generation one, dementia praecox (?), possibly psychosis in a feeble-minded person; in sister, dementia praecox; generation two, feeble-minded plus epilepsy, probable dementia praecox.

No. 96. A three generation family: Generation one, dementia praecox, paranoid; generation two, dementia praecox, paranoid; generation three, dementia praecox, paranoid. This family is unique in that all the individuals are at present in the hospital.

No. 2. Dementia praecox in 11 persons of three generations.

No. 5. Generation one, dementia praecox; generation two, dementia praecox; generation three, dementia praecox.

No. 7. Number C of generation two, dementia praecox; D, of generation three, dementia praecox.

No. 8. Generation one, dementia praecox; generation two, dementia praecox; generation three, dementia praecox.

No. 97. This case is unique in my records since dementia praecox in the ancestor is followed by manic-depressive in a descendant.

No. 98. Generation one, dementia praecox in ancestor; generation two, paranoid psychosis, feeble-minded and dementia praecox.

These 20 families, with but few cases where the diagnosis can be doubted, point very clearly to this conclusion—that dementia praecox breeds true, for it is followed in the great majority of those cases where insanity occurs in the next generation by dementia praecox. In two cases, "moral imbecility" followed. Epilepsy appeared twice in conjunction with dementia praecox psychosis. Very interesting is the advent of mental deficienty. Many of the descendants of these patients are of lower mentality than their parents and there is a distinct trend downward so far as intellectual ability is concerned. Kraepelin's suggestion that many feeble-minded are really congenital dementia praecox patients with dementia as the prominent symptom is valid in my opinion.

Again referring to the recent literature we find that despite the varying interpretations, the above conclusions are completely substantiated. In Rosanoff, of 18 cases of dementia praecox in a parent, 14 cases gave rise to dementia praecox in the descendant, two cases gave rise to manic-depressive, and both of these (Nos. 49 and 72 of his series) present doubtful features. Imbecility and alcoholism follow in two cases.

Krueger relates dementia praecox in the parent to dementia praecox, epilepsy, and imbecility in the descendant. Luther, compiling the cases of Vorster, Kraus, Kriechgrauer, Albrecht, Jolly, and Luther, finds the following (I find his tables correct by personal examination of the works of the writers he quotes); 43 schizophrenic (equivalent to dementia praecox) ancestors with 47 insane descendants of whom

39 were dementia praecox
3 were manic depressive, two of these being Luther's cases, and somewhat doubtful
3 were imbeciles
1 was a psychopathic
1 was an epileptic

Thus the literature bears out this conclusion—dementia praecox in an ancestor is usually followed by dementia praecox in the direct insane descendant. Moral imbecility, feeble-mindedness, and epilepsy are also found in the descendants, and frequently in combination with dementia praecox. Manic-depressive, though related occasionally, seems to be exceptional in the descendants and may represent another hereditary factor at work.

It is interesting to note that Pilcz (155), while agreeing, and, in fact insisting, that dementia praecox is usually followed by dementia praecox, states that in non-catatonic dementia praecox the ancestors show a very large incidence of general paresis and tabes, thus pointing to an injury of the germ-plasm as a starting point for the psychosis. Other writers do not find this statement in regard to neuro-syphilis to be verified in their cases, and I wish to number myself amongst them.

C. THE CASE REGARDING MANIC-MELANCHOLIC STATES (MANIC DEPRESSIVE INSANITY, UNCLASSIFIED DEPRESSIONS)

No. 44. Generation one, typical manic with initial depression and repeated excitements; generation two, manic episode.

No. 45. Generation one, recurrent depression; generation two, dementia praecox.

No. 46. Generation one, agitated depression; generation two, dementia praecox. (Psychosis in the parent somewhat in doubt.)

No. 47. Generation one, depressed episode in psychopathic person; generation two, paranoid presenility, suicidal attempts, depression in two descendants. Other parent also tainted.

No. 48. Generation one, senile depression; generation two, two depressions.

No. 49. Generation one, senile depression; generation two, one doubtful dementia praecox; one chronic depression with paranoid trend.

No 50. Generation one, depression; generation two, chronic depression. Other parent had dementia praecox in her fraternity and in another daughter.

No. 51. Generation one, manic, died in depression; generation two, acute alcoholic hallucinosis.

No. 52. Generation one, manic, depressed, with striking history of manic depressive fraternity and relatives; generation two, manic phase of manic depressive insanity.

No. 53. Generation one, question of manic, question of dementia praecox; generation two, three cases of dementia praecox. Other parent had insanity in his family.

No. 54. Generation one, question of manic question of exhaustion psychosis; generation two, manic depressive insanity.

No. 55. Generation one, depression at involution with a senile psychosis following stroke in fraternity; generation two, catatonic episode in psychopathic person.

No. 56. Generation one, question of manic, probably involutional; generation two, one unclassified short psychosis, one peculiar person, loquacious, and one senile psychosis.

No. 57. Generation one, depression and excitement following childbirth and menopause in two sisters; generation two, involution depression and agitation.

Of these 14 cases, 10 represent what may be termed well established manic depressive insanity. Of these 10, No. 44, No. 48, No. 50, No. 52, No. 53, and No. 54 were followed by manic depressive insanity. No. 51 was followed by acute alcoholic hallucinosis. No. 57 was followed by a psychosis appearing in the involution with perhaps an agitated depression; No. 45 and No. 46 were followed by dementia praecox.

Of the five non-typical cases, one, No. 55, was perhaps late catatonia and was succeeded by dementia praecox but here there was insanity in the other branch of the family. In No. 47, the psychosis concerned a person of peculiar temperament and was

succeeded by psychopathic personality with depression, suicidal attempts and paranoid ideas. Here also there was bilateral taint. In No. 49, a depressed episode at the involution in a peculiar person was followed by a catatonic episode, belonging, undoubtedly, to dementia praecox. In No. 56, the records are scanty in the case of the ancestor.

It will thus be seen that the clean-cut cases of manic depressive are usually followed by manic though also by dementia praecox. As a matter of fact, the diagnosis in case of either psychoses from records is a matter of difficulty and, therefore, the question of relationship of some cases of manic depressive insanity cannot be answered very positively.

Turning now to the literature, we find that for Krueger the relationship is that generally manic-depressive follows manic-depressive but also dementia praecox follows it. Luther, analyzing 62 cases from the records of Vorster, Kraus, Krueger, Jolly, and Luther, shows that of the 77 descendants concerned

	Number of descendant
Manic occurred in	43
Dementia praecox in	22
Idiocy and imbecility in	6
Paranoid condition in	2
Epilepsy in	2
Amentia in	1
Hysteria in	1

We may thus state that the two main trends are to manic and dementia praecox. Thus, for example, Jolly, who opposes dissimilarity in descent, cites 14 cases of manic-depressive in the ancestors, of which four were followed by manic-depressive, eight were followed by dementia praecox, one by paranoia, and one by puerperal insanity which, in my way of thinking, was a dementia praecox. Rosanoff in 10 cases of two generation diseases with manic-depressive in the parent finds four dementia praecox, three manic-depressives, and three epileptics in the descendants. In fact, his belief that manic-depressive is dominant to dementia praecox is based on his findings of dementia praecox in the descendants of manic-depressive. Working from the other direction, that is, from descendant to ancestor on the basis of family history rather than on definite hospital cases, Wolfsohn finds twice as many manic-

melancholic ancestors in the history of dementia praecox patients than of dementia praecox ancestors. It may be stated that all of those authors, excepting Vorster and Sioli, who cite cases in full, give manic-depressive as followed very often by dementia praecox. My own cases lend support to this belief yet I am *not unequivocal in this. I am certain that the clearly uncomplicated manic-depressives are followed by manic-depressives* and that the difficulty of distinguishing some catatonic attacks and some transitory depressions of other diseases from the excitement and depression of manic-depressive cannot be excluded as a factor in the problem.

It may be stated that all the authors agree that idiocy, feeble-mindedness, etc., followed much less closely on manic-depressive than on dementia praecox.

D. THE CASE REGARDING INVOLUTION PSYCHOSES

No. 58. Generation one, involution melancholia or late catatonia; generation two, three catatonic descendants.

No. 59. Generation one, involution melancholia, but terminating in apathy and dementia; generation two, paranoid dementia praecox, two descendants.

No. 60. Generation one, late dementia praecox; generation two, dementia praecox.

No. 61. Generation one, involution melancholia; generation two, dementia praecox.

No. 62. Generation one, unclassified acute psychosis, question of catatonic; generation two, one dementia praecox and one unclassified acute psychosis, question of catatonic.

No. 63. Generation one, late catatonic dementia praecox; generation two, one catatonic dementia praecox, one paranoid dementia praecox.

No. 64. Generation one, acute mania, unclassified; generation two, dementia praecox.

No. 65. Generation one, catatonia; generation two, dementia praecox.

No. 66. Generation one, dementia praecox; generation two, psychopathic personality.

In Group No. 1, four generation family the first ancestor was probably an involution melancholia; dementia praecox in the next three generations.

Of the cases here cited, the psychosis in the great majority of the descendants belonged in the dementia praecox groups. It is, of course, obvious that some of the involution psychoses are nothing but late dementia praecox, but even where this is not the case, termination seems to be the same.

On the question of the psychoses in the descendants of the involution cases there is agreement in the literature. Thus Krueger and Luther, Jolly and Albrecht, representing opposite opinions in so far as similar and dissimilar heredity is concerned, find substantially the same facts; to wit, that the insane descendants of patients with involution melancholia and involution psychoses in general, even aside from those that are diagnosed as late dementia praecox, suffer practically always with dementia praecox.

E. THE CASE CONCERNING ORGANIC BRAIN DISEASE

In 3 of these families, Nos. 67, 68 and 69, the descendants had dementia praecox, but no real good histories had been obtained. In three others, Nos. 70, 71 and 72 such histories have been obtained, and it can be shown that the psychoses in the descendants, involution melancholia and involution depression respectively, can be related to known mental disease in the *other* parent or else to *known mental disease aside from the organic brain disease* of the hospital parent. Therefore, the situation in regard to organic brain disease is not clear. It is very likely that where it is followed by insanity in a direct descendant there are other factors at work.

F. THE CASE REGARDING ALCOHOLIC PSYCHOSES

It is not my intention to emphasize conclusions regarding insane descendants or patients suffering from these diseases—the problem is far too complicated and important to merit anything but the closest study made in a very large number of cases. It is best merely to state that in three of the cases, Nos. 74, 75 and 76, alcoholic psychosis followed alcoholic psychosis. In the other three, Nos. 77, 78 and 79, dementia praecox followed. But other and more directly important psychopathic factors than alcoholism can be shown to exist. In one, No. 77, there is dementia praecox on the other parent's side; in another, No. 78, the alcoholism of the parent was only an incident in the life history of a markedly psychopathic and peculiar person coming of insane stock. In the third, No. 79, the parent shows a remarkable tendency to react to toxic influences of all kinds by severe mental disease.

A very important fact can be added to what is indicated above. If alcoholic abuse is a large factor in inheritable "insanity" then

we should expect it in those racial groups with a large amount of so-called endogenous disease. In the one conspicuous example of a people burdened by such mental disease, the Jews, alcohol is almost unknown. It is true that other factors may play a part in the case of this people, but in any case no work has yet shown that the heavy drinking races are more afflicted with certain mental diseases notably the endogenous ones, than the more temperate racial units.

The case regarding the psychoses of the senium:

No. 80. Generation one, paranoid senile psychosis; generation two, paranoid dementia praecox.

No. 81. Generation one, senile dementia, but with sister with dementia praecox, brother insane, uncle insane; generation two, dementia praecox.

No. 82. Generation one, senile dementia; generation two, alcoholic psychosis. Other parent alcoholic as well as his fraternity.

No. 83. Generation one, senile dementia with a paranoid trend, for years hostile and excited; generation two, catatonic and later paranoid dementia praecox.

No. 84. Generation one, senile psychosis with grandiose and boastful conduct; generation two, an involutional psychosis of grandiose nature.

No. 85. Generation one, senile paranoid state; generation two, senile psychosis.

No. 86. Generation one, senile dementia in an alcoholic; generation two, three descendants insane—one, acute alcoholic psychosis, one, dementia praecox, and one, general paresis.

No. 87. Generation one, senile psychosis not unlike a rapid dementia praecox; generation two, catatonic, question of manic-depressive.

No. 88. Generation one, paranoid psychosis; generation two, dementia praecox plus epilepsy.

No. 89. Generation one, senile dementia; generation two, senile dementia.

No. 90. Generation one, senile dementia; generation two, two epileptics.

No. 91. Generation one, senile dementia; generation two, paranoid condition.

No. 92. Generation one, senile dementia; generation two, dementia praecox.

In addition to these cases we have certain cases already cited. In the paranoid group, families No. 3, 4, and 24, in manic-depressive group, families No. 48 and 49. The senile manics gave rise to manic-depressives. The seven paranoid cases (including the three previously cited) have descendants suffering either from paranoid condition or dementia praecox. Senile dementia without special character is followed by dementia praecox in four cases; senile

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dementia of no special character followed by paranoid condition, one case; senile dementia followed by epilepsy, one case; senile dementia followed by alcoholic psychosis, one case. Unclassified senile psychosis is followed by unclassified involution psychosis, one case. The writers previously quoted, namely, Vorster, (207) Albrecht, Jolly, Luther, and Krueger, all find that in general senile dementia is followed by dementia praecox in the direct descendant. It may be therefore stated that the majority of senile dementia cases, if followed by disease in the descendant, are followed by dementia praecox with a scattering incidence of manic-depressive, paranoid psychoses, epilepsy, imbecility, etc.

If we disregard for the moment the fact that the parent's psychoses were first made manifest at the senium, we find on analyzing many cases that paranoid states breed paranoid states and dementia praecox breeds dementia praecox, imbecility, and possibly epilepsy. Manic breeds manic and perhaps dementia praecox just as happens at any other time of life. There then remains a group of insane persons with a psychosis said to be characteristic of the senium whose insane descendants, according to all the workers, are dementia praecox. These patients, when one discounts the *usual senile deterioration and the usual senile mental attitude*, present a syndrome of apathy at times, changing to excitement at others, destructiveness, hallucinations, delusions, and internal disharmonies, which is nothing less than dementia praecox and which would be so diagnosed did it occur at 30.

CONCLUSIONS CONCERNING THE VERTICAL TRANSMISSION OF CERTAIN MENTAL DISEASES

If then we survey the facts here presented together with those ascertained by a survey of the literature we find:

1. That the paranoid diseases tend to paranoid states, perhaps finally to dementia praecox states.

2. That the manic-melancholic diseases are in the main followed by manic melancholic diseases, but in a certain number, especially of doubtful cases by dementia praecox.

This brings up the important point that in several of the cases presented in this book patients have gone through repeated attacks of what appear to be manic-depressive insanity to wind up with what appears to be dementia praecox. Of late years many have

pointed out that catatonic states occur in manic-depressive insanity. It seems to me very probable on the basis of individual cases and the history of family mental disease that catatonic states are an intermediary form of mental disease, bridging the gap between manic-depressive insanity and dementia praecox.

3. That the involutional and senile state if paranoid, trend towards paranoid states and dementia praecox.

4. That the manic-depressive states of involution and senium trend towards manic-depressive and dementia praecox, especially the latter.

5. That dementia praecox in an ancestor trends towards dementia praecox in the descendants with a certain scattering incidence of imbecility. This imbecility seems to me to be in part at least of the Kraepelinian congenital or very early dementia praecox.

6. Neither for organic disease or alcoholic disease can anything definite be said. Wherever good histories are obtained other and more definite psychopathic factors are found.

"It will thus be seen that all roads seem to lead to dementia praecox and from thence to imbecility." Quoting from my own (140) publications I find it over-emphasizes the drift of things from generation to generation. Paranoid characters remain very persistent, and so do manic-depressive characters even though a certain number of cases follow this drift. This may be stated, that when the disease gets worse from generation to generation it ends in dementia praecox, but this is not always the case and there must be a huge number of mild cases of mental disease in the descendants of the insane, who represent an upward trend, a recovery trend. Rare indeed is that mingling of stocks whereby a mental disease persists unaltered for more than two generations. As has been pointed out in a previous chapter the earlier onset of the disease and its worsening preclude propagation, for while the feeble-minded *may* have many illegitimate children dementia praecox patients do have less children than the normal population and as their marriage rate is low there finally comes a point in the history of many, perhaps of most psychiatric families when the stock dies out.

CHAPTER XI

Horizontal Transmission of Mental Disease

1. Given two or more insane siblings, do their psychoses tend to be like or unlike? If like, what diseases are most part to run through such a fraternity? If unlike, are there any disease groups that are mutually exclusive?

2. This brings up another question which bears upon the relationship of the two great, so-called endogenous mental diseases—dementia praecox and manic-depressive insanity. In the previous chapters it was stated that manic-depressisve in an ancestor was quite likely to be followed by dementia praecox in a direct descendant, but that the reverse, dementia praecox in an ancestor and manic-depressive in a direct descendant occurred but seldom, if at all. If this be so, how about the occurrence of the two diseases in the same fraternity? Does that happen commonly or exceptionally? The evidence, one way or another, has been gathered from the literature and from the Taunton cases.

It needs again be emphasized that very grave difficulties present themselves at the very outset of the study, and in order not to incur the reproach of having overlooked them, I shall state them in some detail. In the first place, psychiatric diagnosis, except in a few diseases, is empirical and rests on no sure foundation. The classification is not biological and originally was based on criteria that have been largely abandoned, so that it is now approaching a haphazard state again. Once, we believed that dementia as an end result in dementia praecox was a terminus that established the diagnosis—this has long since been abandoned as a *necessary* sign. At the same time we were told that a circular course denoted manic-depressive insanity, especially if there was a free interim. But now, the accepted authority Kraepelin has a place for circular dementia praecox. Catatonia stepped from its high place as a disease entity and became a symptom of dementia praecox, but the flight of time has brought a group of observers to the point where they speak of it as a symptom of manic-depressive insanity while one writer, Urstein, has re-elevated it to a disease in itself. This is not all nor yet the worst, for the foundations of psychiatry are being

crumbled by the attacks of psychoanalysts who claim to find mechanisms at work which, if accepted, render puerile classification based on more superficial characters. Schizophrenia is now the magic word which explains very diverse appearing conditions, and it is a commentary on the eager desire for a solid basis upon which to rest our diagnoses and our conceptions of mental diseases that this term is rapidly supplanting dementia praecox in many clinics.

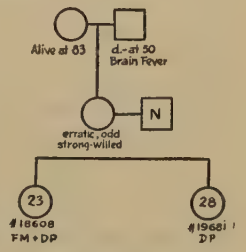
Add to these inherent difficulties the misleading character of records as a basis for a diagnosis and the task assumes a forbidding aspect. *If it is so difficult in life to differentiate between a stuporous depression and a catatonic stupor, how can one do so from a record, perhaps written by a bored, uncritical, junior physician whose mind was decidedly elsewhere or an executive official absorbed in cutting down expenses?*

A happier side of the situation presents itself. The great majority of dementia praecox cases in their asylum aspects are clear-cut. Any group of psychiatrists walking through the wards of an insane hospital will agree without question on the majority of cases that they see. The cases that merge with manic-depressive are a small though compact group, and the questions that arise in differential diagnosis in such cases are apt to be one of the following: Is this an excited catatonic or an excited manic? Is this a stuporous depression or a catatonic stupor? The only way out of the difficulty in the doubtful cases is not to stick too closely to the question, "Is this manic, dementia praecox, or what not?" One may ask of two cases, "Are these essentially similar from the biological standpoint, do they present agreement or difference?" and much of the difficulty of finding a name evaporates.

Part I. Brothers and Sisters

Sib Group No. 1 (Dementia Praecox in All Members)

A. Female, No. 18608, single, 23 years on entrance March 5, 1909. Maternal grandfather, "brain fever," age 50. Maternal grandmother alive at 83. Mother erratic, odd, strong-willed, dominant, extravagant. Father normal. Patient did poorly in school, without doubt feeble-minded, poor physically. In hospital, mute, had to be fed, dirty,



Group No. 1

masturbated. Became demented, untidy, talked in disconnected manner. In hospital now.

B. Female, No. 19681, single, 28 years on entrance June 7, 1911. Onset with excitement, destructiveness, flightiness, active auditory and visual hallucinations, was disturbed, hallucinated, destructive, obscene, negativistic. Discharged unimproved to private hospital October 30, 1912.

SIB GROUP NO. 2

A. Female, No. 14615, single, 22 years on entrance June 4, 1900. Father had epileptic attacks. Onset with confusion, paranoid delusions of vague character, hallucinated, mute, refused food. In hospital, reticent, seclusive, occasionally violent, demented. Transferred to Medfield January 5, 1904.

B. Female, No. 16175, single, 23 years on entrance October 28, 1903. Onset at 19. It is now stated paternal uncle was insane. Patient had queer spells for years. In hospital, negativistic, mute, demented, occasionally violent. Transferred to Medfield.

C. Female, No. 18688, single, 25 years on entrance May 15, 1909. Had an illegitimate child. Onset with excitement, talkativeness, later became hallucinated. She became demented, curled upon bench, obscene, hallucinated and seclusive. Transferred to Medfield.

SIB GROUP NO. 3

A. Female, 14639, married, 26 years on entrance July 2, 1900. Father died of tuberculosis. Was a bright, vivacious girl, practiced self-abuse for years. Depressed at 17. Eleven months after marriage had child. Then became profane, obscene, incorrigible. Was sent to a sanitarium. Gradually grew worse. In hospital, restless, disturbed, had to be restrained, grew demented, hallucinated. Died with pulmonary symptoms, probably pulmonary tuberculosis, June 9, 1901.

B. Male, No. 20306, separated from wife, 35 years on entrance August 20, 1912. Backward in studies, seclusive, masturbation. Had gonorrhoea in 1906. Delusions of persecution, hallucinations of hearing, ideas of reference marked, apprehensive. Short improvement for time, became suicidal as a result of auditory hallucinations. Became depressed, apathetic and seclusive, demented, active hallucinations. Died of pulmonary tuberculosis July 10, 1916.

Note. The sister is said to have been bright, the brother always dull, yet both individuals developed very similar psychoses and died of pulmonary tuberculosis.

SIB GROUP NO. 4

A. Male, No. 15898, 30 years on entrance September 15, 1898. Onset a year before admission. Auditory hallucinations, marked insomnia. On

admission active auditory hallucinations, delusions of persecution and reference, impaired memory, suspicious, unsociable, threatening and violent at times. Later became demented, apathetic and untidy.

B. Female, No. 20329, single, 21 years on entrance September 4, 1912. Father died of an apoplectic attack. Patient one of 13 children, five died of "meningeal" condition. There are five living sisters, all are rather frail. Brother, above patient. Very backward in school. At the age of 14 left in the fourth grade. Always quiet, had severe headaches. Auditory hallucinations commenced at 17. Untidy, restless, insomnia, memory became impaired. In hospital, restless, hallucinated, silly, disoriented, delusions of persecution. In hospital at present, demented, untidy, occasionally mutters to herself. Answers are unintelligible.

SIB GROUP NO. 5

A. Female, No. 18029, single, 22 years on entrance December 5, 1907. Father was always somewhat slow of comprehension, considered defective. Patient was always somewhat backward, left school at 17 in the fifth grade. In hospital, was restless, active, auditory and visual hallucinations, was confused, irrelevant, disoriented, untidy in habits, became very much demented. In hospital at present.

B. Female, No. 18118, single, 20 years on entrance January 29, 1908. Was committed to Waverly School for Feeble-Minded December 6, 1907. Seclusive, never talked much, never strong, never menstruated. In Waverly, slow about work, forgetful, delusions of persecution. In this hospital, intractable, noisy, delusions of persecution, resistive, fabrications, unstable emotionally, apathetic and indolent. Later she became very silly, showing increasing dementia and auditory hallucinations. Died of acute pulmonary tuberculosis February 13, 1914.

SIB GROUP NO. 6

A. Male, No. 17279, single, 25 years on entrance April 18, 1906. At home, depressed, deluded, hallucinations of hearing. No further history. In hospital, depressed, unstable emotionally, deluded, talkative and noisy. Active auditory hallucinations and persecutory ideas, demented. Transferred to Worcester State Hospital, June 10, 1908.

B. Female, No. 20640, married, 24 years on entrance March 29, 1913. Nothing of note in early history, sociable and congenial. Married at 21, three children. After first child had attack like present. This illness began three or four weeks before admission. Thought people talked about her, sleepless, ideas of infidelity. In hospital, confused, later memory became good, orientation perfect, agitated, noisy, apprehensive, active auditory hallucinations. Later, somewhat improved. Discharged December 14, 1914, diagnosis, dementia praecox.

SIB GROUP NO. 7

A. Male, No. 13673, single, 32 years on entrance January 2, 1898. Had three previous attacks. In Taunton, Danvers and Medfield hospitals. Delusions of persecution, irritable, has pressure in the head, buzzing in the ears, sees spirits in room. In this hospital developed many eccentricities, knelt and prayed a good deal, seclusive, hears the devil speaking to him, moderately demented, industrious. At present in hospital, a good worker, markedly hallucinated, shows many eccentricities, moderate delusions of persecution.

B. Female, committed to this hospital on six different occasions. First commitment, No. 15001, May 29, 1901; last commitment, No. 22588. Erotic, believes that she is pregnant and has so believed for six years. Believes that the nails of Christ are on her hand. Considerable motor activity, has delusions of persecution and active auditory hallucinations, slight dementia.

SIB GROUP NO. 8

A. Male, No. 12100, single, 37 years on entrance January 17, 1894. Delusions of persecution, hallucinations of sight and hearing, threatened violence, disoriented, suspicious, reticent, restless, hallucinated and demented. In hospital at present.

B. Female, No. 17276, single, 36 years on entrance April 14, 1906. No history. In hospital, demented, careless about exposure of body, untidy, apathetic, reacts to hallucinations of hearing, indolent and demented. Discharged to Medfield.

SIB GROUP NO. 9

A. Male, No. 16340, single, 29 years on entrance June 6, 1904. Paternal side negative. Maternal aunt and maternal cousin insane. Mother died suddenly. Always very backward. Hallucinations of sight and hearing marked, delusions of persecution, ideas of reference, very incoherent. In hospital at present, very demented, negativistic, untidy, salad speech, marked apathy.

B. Male, No. 16434, single, 26 years on entrance March 29, 1904. Symptoms exactly as brother.

SIB GROUP NO. 10

A. Male, No. 16066, single, 29 years on entrance August 12, 1903. Apathetic, deluded, very hallucinated, demented and restless. Transferred to Worcester.

B. Male, No. 16073, single, 31 years on entrance August 20, 1903. Father said to have been insane for short period from 24th to 26th year. Patient excited at 26. Believed spirit had come to him and given him great power, delusions of grandeur and persecution very marked, sexual ideas prominent,

later developed religious ideas of grandeur, tried to marry several unknown women, wrote erotic letters, was oriented, memory fair, somatic ideas prominent, seclusive and apathetic. Hallucinations of sight and hearing. Transferred to Worcester, paranoid dementia praecox, possibly paranoia.

SIB GROUP NO. 11

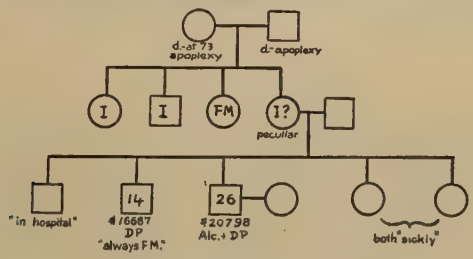
A. Male, No. 10174, single, 31 years on entrance September 26, 1887. On admission incoherent, demented, mute, probable hallucinations of hearing, resistive and cataleptic. Tube fed. In hospital, became excited, had epileptic attacks following which he was talkative, became demented, incoherent, occasionally violent. Died January 3, 1890, of tuberculosis.

B. Female, No. 15922, single, 30 years on entrance May 4, 1903. Commitment papers stated, "Hallucinated, suicidal tendencies, depressed, nervous." Did very poorly in school. Three weeks before admission attempted suicide by jumping into the river. In hospital, partly disoriented, stupid, slovenly, untidy in habits, a few delusions, incoherent in speech, wandered about the ward aimlessly. Question in this case of general paralysis, absent knee-jerks and ankle-jerks.

Note. There is a question whether B is not a general paretic.

SIB GROUP NO. 12

A. Male, No. 16687, single, 14 years on entrance December 22, 1904. Maternal grandmother died at 73 of apoplexy. Maternal grandfather died of apoplexy. Mother is peculiar, believed to be insane. One maternal aunt



GROUP NO. 12

insane, also maternal uncle. One maternal aunt feeble-minded. One older brother of patient in hospital, two younger sisters sickly. Early history, dentition convulsions, had much sickness, *always feeble-minded.* Markedly nagativistic, takes attitudes and maintains them for long time, destructive, very much demented, exceedingly violent at times, mute. In hospital at present.

B. Male, No. 20798, married, 26 years on entrance July 9, 1913. Graduate in dentistry, always heavy drinker. Said to have diabetes. Assaulted his wife, excited, delusions of jealousy, obstinate and haughty, delusions of

grandeur marked. He is greater than anybody else, Saint Victor and King of the Jews. Hyperreligious at times, hears the Virgin Mary, many mannerisms, loose, incoherent speech, apathetic and shows gross sexual ideas. In hospital at present.

SIB GROUP NO. 13

A. Female, No. 15452, single, 21 years on entrance May 23, 1902. Family history is negative. Patient always very backward in school and otherwise. In 1902, hallucinations of sight and hearing, became dirty and demented. In hospital, apathetic, demented, bench type. Sent to Medfield June 5, 1904.

B. Male, No. 21591, single, 28 years on entrance August 28, 1914. Marked delusions of persecution and reference, hallucinations of hearing, demented. Transferred to Medfield.

SIB GROUP NO. 14

A. Male, No. 14177, married, 46 years on entrance May 27, 1899. Onset for years. Delusions of persecution and reference, excited, suspicious, coherent. Diagnosed as paranoid psychosis. Transferred to Medfield.

B. Female, No. 16508, single, 58 years on entrance August 4, 1904. Second commitment, No. 18580. Coherent, memory O.K., delusions of persecution and reference, vague, fleeting hallucinations, marked lack of initiative, thought the women nurses in the hospital were men in disguise. Tube fed for a time. On re-entrance was irrelevant, incoherent, unstable. Died of organic brain disease. Paranoid psychosis is perhaps as far as one could venture on the data given.

SIB GROUP NO. 15

A. Female, No. 17152, single, 50 years on entrance January 8, 1906. Always backward in school, feeble-minded. She and her sister, who entered at the same time, shared delusions in common. Marked ideas of persecution, irritable, fantastic, hallucinations of sight and hearing. In hospital, became quiet and industrious, a good worker. At present in hospital, demented, apathetic.

B. Female, No. 17153, single, 31 years on entrance January 8, 1906. Always simple. Delusions of persecution of vague type, incoherent in speech, rambling, frequently aggressive, hallucinations of sight and hearing, delusions of poisoning marked. At present in hospital, demented.

SIB GROUP NO. 16

A. Male, No. 15065, single, 16 years on entrance January 4, 1901. Stomach and bowels are all tied up, nothing can pass through him, some influence is on him, marked mannerisms, seclusive, memory very poor, demented and silly. In hospital at present.

B. Male, No. 16312 and No. 16474, single, 21 on first admission. Onset at 19. Very stubborn, delusions of persecution, sexual delusions and reference very marked, hallucinations. In hospital, became disoriented and demented. At present in hospital. Diagnosis, dementia praecox.

SIB GROUP NO. 17

A. Female, No. 15256 and No. 16511, single, 29 years on first entrance November 30, 1901. Much insanity on both sides of the family. At 25 was admitted to a hospital in Dublin, Ireland. Had then been sick for two years. Claimed she was mesmerized, the evil one was influencing her, had exalted religious ideas, thought she was the Virgin, marked somatic delusions. In this hospital became very obscene, mannerisms, stereotypy of an obscene kind very prominent symptom, negativism marked, delusions of persecution, ideas of grandeur vaguely expressed. In restraint most of the time since entrance until December, 1915, when she sustained a hemiplegic stroke and since then has been quiet though exceedingly deluded, obscene and demented.

B. Male, No. 17794, single, 24 years on entrance May 31, 1907. Had tuberculosis of the hip. Always feeble-minded. In hospital, dull, stupid, became mute, had to be tube fed. At other times very much excited, demented. Died of tuberculosis January 25, 1908.

C. Male, No. 13691, single, 18 years on entrance. Onset at 16. Has become quiet, apathetic, deluded. In hospital, "memory impaired, active delusions of persecution, hallucinations of hearing and sight, tends to be mute." Became apathetic and exceedingly demented. In hospital at present. An uncle is also dementia praecox.

SIB GROUP NO. 18

A. Female, No. 14970, single, 30 years on entrance April 29, 1901. Commitment papers state, "Says she is to have a baby. Laughs continually, has auditory hallucinations, talks in a halting manner." No history. Filthy on admission, had to be tube fed, thought there was poison in the food, episodes of causeless laughter, violent at times, demented. Discharged August 5, 1901, for deportation to Canada.

B. Female, No. 14971, single, 30 years on entrance April 29, 1901. Says she is to hanged for some crime she has committed. Could not sleep. She thought people would get in from the windows. Kept curtains down and windows locked for fear some one would do her bodily harm. Sits in window greater part of day, saying she is sick. In Hospital, talks in foolish manner, takes little interest in work, looks vacantly about her when not occupied. Discharged to State Board of Insanity for deportation to Canada.

Note. In the above cases though diagnosis of dementia praecox is made it is evident that there is considerable room for doubt. Possibly a longer observation would have shown reasons for a different diagnosis.

SIB GROUP NO. 19

A. Male, No. 18039, single, 28 years on entrance December 10, 1907. Family history negative. Alcoholic, unsteady worker. In hospital, very negativistic, will not talk, hallucinated, filthy. In March, 1908, still negativistic. June 12, 1908, improved somewhat and then discharged.

B. Male, No. 20581, single, 37 years on entrance February 18, 1913. It is now stated two maternal aunts were insane, also an uncle was a drinker for one half years. Has been hyperreligions, believes that sounds come to him and that his head is charged with electricity, hallucinations of sight and hearing. In hospital at present, shows many mannerisms, religious delusions, silly facial expression and conduct, demented, occasionally violent.

Note. First patient had catatonic attack. Second case shows dementia praecox with paranoid symptoms.

SIB GROUP NO. 20

A. Female, No. 16858, single, 43 years on entrance May 4, 1905. Grandparents said to have died of old age. Eight brothers and sisters, of whom one is dead. Ordinarily bright, always a leader. In 1904, "father and stepmother came out with cats heads on. Later on they had dogs heads. Came and looked in through window, also came in as cows. People say she and her sister will have babies. Many ideas of persecution on the part of neighbors, ideas of reference." Discharged, boarding-out patient.

B. Female, No. 16859, single, 58 years on entrance May 4, 1905. Same symptoms as sister. Discharged to the State Board of Insanity June 30, 1905, boarding out.

SIB GROUP NO. 21

A. Male, No. 16352, single, 24 years on entrance January 10, 1908. Father died at 48 after having been insane for four or five years. Mother died at 52, disease of the heart. Sister now living, had paralytic stroke. Worried over death of sister, was irritable, sleepless, prayed a good deal, held conversation with imaginary persons at night, thought that "he was the divine gift of heaven." In hospital, quiet, talked smilingly of hallucinations and delusions, ideas of religious grandeur marked. Became restless, cross, muttered a good deal, showed cerea flexibilitas. At times impulsive, became demented. Transferred to Medfield State Hospital, March 26, 1909.

B. Male, No. 16352, 34 years on entrance April 9, 1901. It is now learned that father died at Medfield Insane Hospital of dementia praecox. Always melancholic in disposition, became dull, stupid, disoriented, confused, marked delusions, sits in one posture for long time, believes he is Almighty God. Became demented, occasionally aggressive. Transferred to Medfield.

SIB GROUP NO. 22

A. Male, No. 13043, single, 20 years on entrance January 13, 1899. Mother had been insane. Stole a razor, became violent, had ideas of persecution. In hospital, became depressed, demented, hallucinated. Died of tuberculosis March 13, 1909.

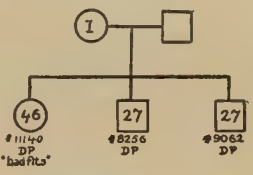
B. Female, No. 14812 and No. 15254, 28 years on entrance December 1900. Has hallucinations of hearing, delusions of persecution and reference, delusions of jealousy. Moderately demented.

SIB GROUP NO. 23

A. Male, No. 9062, single, 27 years on entrance December 27, 1883. Wild, rambling in comversation, threatened his mother. In hospital, sits around quietly, will not answer questions, keeps head bowed, never talks, acts demented. Transferred June 9, 1886, without showing much change.

B. Male, No. 8256, single, 27 years on entrance February 21, 1881. Was demented on entrance. Died November 13, 1882.

C. Female, No. 11140, single, 46 years on entrance June 27, 1891. Stated that mother was insane. Patient has been insane for many years, is now dangerous, demented, memory poor, fault-finding. Has had fits from the age of 7 to 14. Discharged April 11, 1892, to town authorities.



GROUP NO. 23

Note. Though the records are scanty in the case of each, the onset and course of disease point almost unmistakably to praecox.

SIB GROUP NO. 24

A. Female, No. 14438, single, 34 years on entrance December 18, 1899. Ideas of grandeur, going to inherit a lot of money, prayed a good deal. In hospital became markedly demented and muttered to herself. Transferred to Medfield December 3, 1907, unimproved.

B. Male, No. 14430, single, 36 years on entrance December 8, 1899. Had millions of dollars, owned all the houses, walls and floors of hospital made of pearls and diamonds, memory was good, oriented. At first good-natured, noisy. Later, hallucinations of hearing, broke windows because people outside were calling him bad names, noisy, destructive, profane, became quarrelsome and irritable, gradually quieted down, did not work, demented. Transferred to Medfield State Hospital, not improved, January 12, 1903.

C. Female, No. 17759, single, 30 years on entrance April 29, 1907. Record very scanty, "is euphoric, has delusions of religious nature, grandiose." Nothing more stated. Diagnosis is given as pranaoia.

Note. In this family there is a striking similarity of psychotic type. All had grandiose ideas, ideas of religious character, they gradually became quieter and demented moderately.

SIB GROUP NO. 25

A. Male, No. 7809, single, 37 years on entrance November 25, 1878. Insane heredity on mother's side. Is violent, dangerous, calls himself Christ. Was discharged as recovered. Re-entered, No. 8292, 46 years, March 29, 1881. No hallucinations, coherent, good workman, orderly, clean, believes he is a prophet, God speaks through him. Sleeps on the coldest nights with only a sheet over him. Eloped February 17, 1888. Throughout stay maintained delusions, was quiet, orderly, non-demented.

B. Female, No. 8805, single, 30 years on entrance March 18, 1883. Had a previous attack after typhoid and recovered. On entrance violent and maniacal, noisy, incoherent, destructive, became seclusive, lay on floor in doorway, had to be led to meals, never spoke. In 1887, it is stated that she was incoherent, destructive, apathetic, muttering and demented. Died June 18, 1891 with a diagnosis of dementia praecox, catatonic.

Note. The brother in this family showed marked paranoid ideas which persisted over years and were considered by the diagnosticians of the time as paranoia. The sister suffered from undeniable dementia praecox. Whether the brother's psychosis belongs in paranoid dementia praecox or not is difficult to state from the records.

SIB GROUP NO. 26

A. Male, No. 12379, single, 16 years on entrance November 5, 1894. Mother melancholic for the last two years of life. Father alcoholic. In hospital, masturbates, noisy, incoherent, usually does not answer questions, hallucinations of sight and hearing became prominent, at times stuporous. Died April 8, 1897.

B. Male, No. 12573, single, 15 years on entrance October 15, 1895. Excited, incoherent, destructive. Grew very quiet, seclusive, demented. Transferred to Medfield May 1, 1896.

C. Male, No. 13509, single, 13 years on entrance July 21, 1897. Violent, noisy, quarrelsome. Never went to school. Has been insane at times for 18 months. In hospital, became quiet, tractable, very demented. Transferred to Medfield October 1898.

Note. Each of these three patients was recorded as feeble-minded throughout life. The psychosis that developed on this basis seems to me undeniably praecox, and it is likely that the patients were dementia praecox.

SIB GROUP NO. 27

A. Female, No. 16317, single, 27 years on entrance February 29, 1904. Father heavy drinker. Had scrofula in early life. Three weeks before admission developed religious ideas, thought she had been a wicked woman, that there was a devil in her, heard voices telling her that she was to die. Became destructive and refused to eat, had auditory and visual hallucina-

tions, memory good, orientation good, insight lacking, negativistic, apprehensive. Died March 11, 1904, of exhaustion.

B. Male, No. 18746, single, 35 years on entrance February 5, 1905. Second admission June 25, 1909. During the first stay in hospital was dull, confused, suspicious, destructive. Improved slightly and was discharged September 29, 1906. On second admission memory was fair, confused at times, auditory hallucinations. Frequently had period of marked irritability and resistiveness. Later became disoriented as to time and place, auditory hallucinations were prominent, became apathetic, flow of thought decidedly retarded, became seclusive and demented. At present in hospital.

SIB GROUP NO. 28

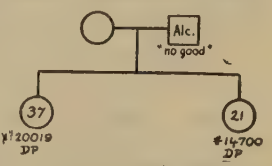
A. Male, No. 16594, single, 19 years on entrance October 13, 1904. Typical dementia praecox. Hallucinations of sight and hearing, mute, seclusive, resistive, became demented. Died of tuberculosis September 10, 1906. Autopsied.

B. Female, No. 18884, single, 22 years on entrance September 20, 1909. It is stated now that father had severe headaches for several years and did not work. Patient is very devout. Psychosis started when she commenced to visit priests and accuse them of immoral conduct with nuns. Despite the fact that she claimed to be a devout Catholic she denied the existence of God, the devil and the church. Said a young man was hypnotizing her for sexual purposes. Ideas of hypnosis and reference very marked. In hospital, became very negativistic and seclusive, hallucinated and demented. Discharged to Medfield, not improved, May 8, 1911.

SIB GROUP NO. 29

A. Female, No. 14700, single, 21 years on entrance August 17, 1900. Assumed prayerful attitudes continually, will not answer questions, masturbates, untidy and demented. Was discharged to Medfield November 21, 1901.

B. Female, No. 20019, single, 37 years on entrance May 7, 1912. Father is a chronic alcoholic and "no good." Seclusive and proud in early life. Left school at the age of 12. Developed exceedingly prominent delusions of persecution and reference. In the hospital, showed hallucinations of hearing, said evil influences were playing on her. In hospital at the present time, demented, negativistic, hallucinated.

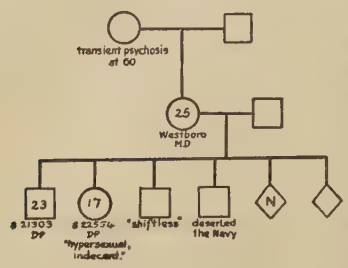


GROUP NO. 29

SIB GROUP NO. 30

A. Female, No. 22554, single 17 years on entrance June 14, 1916. Family history in this case is exceedingly interesting. (See chart.) The maternal grandmother had a transient psychosis after grippe at the age of 60. Her

daughter, the mother of the patients of the generation to be considered, was in Westboro State Hospital at the age of 25 with a psychosis that was diagnosed as manic-depressive insanity and appears to have been of that character. The paternal side entirely negative. There were six descendants, two of whom came to the hospital. Of the four who did not come, one was a sailor, and said to be shiftless. Another was a sailor, deserted from the navy, and whereabouts unknown. One person said to be normal. The other is at present a child. This patient entered the hospital excited and talkative, delusions of poisoning were very marked, erotic in her conduct, "hypersexual indecent." In hospital, markedly deluded, mutters to herself, filthy, very destructive, demented, seclusive, except when very excited. In hospital at present.



GROUP NO. 30

B. Male, No. 21303, single, 23 years on entrance April 13, 1914. Said to be backward in school, seclusive in conduct throughout life, developed marked incoherent delusions of persecution. In hospital, resistive, negativistic, seclusive, marked delusions of persecution, dementing.

SIB GROUP NO. 31

A. Female, No. 14746, married, 35 years on entrance October 17, 1900. Said to have been melancholic and to have had bad temper. Was in insane hospital in London, England, for three and half years. This psychosis started with insomnia, quarrelsomeness, destructiveness. In hospital, memory poor, very deluded, auditory hallucinations, noisy, quarrelsome, destructive and violent, demented. No change during the two years of her stay in hospital. Discharged to Medfield October 16, 1902. Thought to have been imbecile from start.

B. Female, No. 22137, married, 36 years August 3, 1915. Had a previous admission January 8, 1912, to June 29, 1914. Early history negative. This attack commenced with headache, rambling conversation, insomnia and increased religious fervor. Refused to eat, became melancholy, later was noisy and incoherent, distinct delusions of persecution and poisoning, memory was good, orientation perfect, grasp on surroundings firm, delusions of persecution moderate, hallucinations of sight and hearing, showed dementia. Was transferred to Grafton September 20, 1916.

Note. The record in this case gives some doubt as to the diagnosis of praecox, but report from Grafton and statements of physicians who have seen patient leave no doubt that she is praecox at the present time.

SIB GROUP NO. 32

A. Female, No. 18327, single, 30 years on entrance June 22, 1908. Has always been backward in school and general work. Considered feeble-

minded. Is excited, takes fixed attitudes, and holds them for hours at a time. Generally apathetic, entirely mute. Remained so throughout most of her stay in the hospital. Was discharged to Medfield where a similar diagnosis has been made.

B. Male, No. 2207, single, 23 years on entrance October 21, 1915. It is now stated that three of the fraternity were insane, but details are lacking. Patient has been a vagrant and a thief. Always somewhat feeble-minded. Has active auditory hallucinations, shows marked impulses, delusions of poisoning are prominent, became silly and demented. At present in hospital, shows verbal salad (incoherent speech), undidiness, dementia, vague hallucinations and delusions.

The following cases belonging to the same group, that is, where brothers and sisters show consistent dementia praecox, have been previously detailed in chapters on Vertical Transmission of Mental Disease. These previous chapters dealt with them from the standpoint of a psychosis presented in several generations. Since this chapter deals with the psychoses presented by members of the same generation, I shall but briefly refer to the families. For details, see the previous chapters.

In order to save space and labor I shall first give the number of the family group as it occurs in the sibling series and in parenthesis the number as it occurs in the previous chapters.

SIB GROUP NO. 33 (FAMILY GROUP NO. 22)

C, D and E (two males and one female). Condition typical in each case. History, that there are three feeble-minded brothers and sisters. Two first cousins of the above were also in the hospitals with the diagnosis, dementia praecox. The disease in the ancestors was dementia praecox.

SIB GROUP NO. 34 (FAMILY GROUP NO. 13)

B and C (two males). Dementia praecox in each with feeble-mindedness in C. The ancestors showed paranoid disease, probably paranoid dementia praecox.

SIB GROUP NO. 35 (FAMILY GROUP NO. 27)

B, C and D (One female and two males). The two males were twins. The female showed paranoid dementia praecox, the brothers hebephrenic dementia praecox. The mother, who was also in the hospital, showed paranoid condition, perhaps paranoid dementia praecox.

SIB GROUP NO. 36 (FAMILY GROUP NO. 29)

There were present in this hospital of this group five members of the same generation. In one case, A, manic-depressive insanity may be considered as a diagnosis, though the records are scanty. In all the others, the diagnosis of the hospital staff was dementia praecox. In general, the condition was periodic, paranoid, with ideas of grandeur, religious delusions, fixed attitudes and mannerisms, hallucinations of sight and hearing. Dementia is not a prominent symptom in the family. A son of one of the members, B, is in the hospital now with a similar psychosis which is diagnosed dementia praecox. There were several other members of the family all of whom were peculiar and had the same general disposition; that is, they were suspicious, had grandiose ideas, were continually in trouble and claimed divine powers.

SIB GROUP NO. 37 (FAMILY GROUP NO. 37)

A, B, C and D (three females and one male). Each presented similar psychoses, excitable, hallucinated, irritable, demented. One of these, A, had a son, C, who showed feeble-mindedness and epilepsy.

SIB GROUP NO. 38 (FAMILY GROUP NO. 39)

A and B of this series (two females). Characteristic symptoms of dementing type of dementia praecox. One of these, A, had a daughter, C, who is now in the hospital and is considered typical hebephrenic dementia praecox.

SIB GROUP NO. 39 (FAMILY GROUP NO. 40)

B, C and D (two males and one female). Presenting similar psychoses, very demented at the present time. The mother of these patients was in this hospital with dementia praecox.

SIB GROUP NO. 40 (FAMILY GROUP NO. 43)

A and B (male and female). Brother's condition was intermittent. The sister's was more stationary. The male had a daughter who entered this hospital and is here now with a diagnosis of feeble-mindedness plus epilepsy.

SIB GROUP NO. 41 (FAMILY GROUP NO. 53)

B, C and D (two females and one male). At present in the hospital. Psychoses very similar. Paranoid ideas, hallucinations, violent, aggressive conduct and dementia, usually apathetic and indifferent. The mother was in this hospital with a psychosis that was diagnosed as manic-depressive insanity, may, however, have been late catatonia.

SIB GROUP NO. 42 (FAMILY GROUP NO. 58)

B, C and D (two females and one male). As is stated in the previous chapters, the diagnosis here is decidedly in doubt. It is possible that an agitated form of manic insanity was the psychosis. There is a very striking similarity in the course of the condition in each of the siblings, ending in death within two weeks of entrance to the hospital in each case and having its onset at about the same period of early middle life.

SIB GROUP NO. 43 (FAMILY GROUP NO. 59)

B and C (male and female). Both somewhat feeble-minded. Dementia praecox, paranoid form, properly diagnosed in each case. Both transferred to Medfield State Hospital. The mother of the patient was in this hospital with dementia praecox or involution psychosis. There was, however, insanity on both sides of the family.

SIB GROUP NO. 44 (FAMILY GROUP NO. 93)

Two groups of brothers and sisters are here represented, the one belonging in the first generation and the second, descendants of one of these. A and B show psychoses strongly resembling agitated depression and involution melancholia. Two daughters of the first, C and D, are at present in the hospital; dementia praecox in one and dementia praecox, paranoid, in the other. There is a striking difference in the two conditions; in the first dementia very prominent, in the second none. Tuberculosis in D.

SIB GROUP NO. 45 (FAMILY GROUP NO. 95)

A and B (two males). Mild paranoid dementia praecox in one; feeble-mindedness with hebephrenic dementia praecox in the second.

As stated before, the above constitute those cases of dementia praecox in siblings in which the question of differential diagnosis did not enter. That is to say, practically all the observers concerned united in the diagnoses. Later on, cases will be detailed in which the diagnosis is more difficult, but in which it is believed that dementia praecox is the proper caption.

In the group which is to follow, manic-depressive is the diagnosis made for all those members of the family who came to the hospital.

SIB GROUP NO. 46

A. Female, No. 15266, married, 58 years on entrance December 7, 1901. Throughout life has been somewhat easily depressed and excited, and has been either depressed or excited for short periods of the time. At meno-

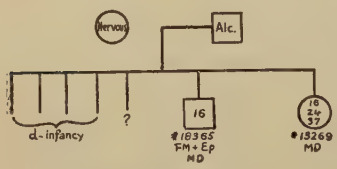
pause, showed much irritability, lowered efficiency, and depressed mood. Recovered. One sister was insane for a year. Recovered. One brother is an alcoholic. Onset, got up in the night to do her washing, and made foolish purchases, became sleepless, would not stay in the house. In the hospital, rambling, flighty and euphoric, no hallucinations, excited and restless, interfering with other patients and giving her advice freely on how to run the hospital. After a year in the hospital became better and was discharged May 31, 1902. At home has been fairly well. It is stated, however, that her capacity for work has been lowered.

B. Female, No. 15900, single, 58 years on entrance April 14, 1903. At 46 became peculiar, gave up position in hotel because of forgetfulness. Began to get run-down and became excited. Was in McLean Hospital April 8, 1891. Slept poorly, became talkative and incoherent, excited and restless, became exhilarated. In June, was despondent. In September, was discharged much improved. Remained well in interim. Entered Taunton State Hospital after an attack of grip. Was talkative, noisy and excited for a short period, often unreasonable, no hallucinations, no definite delusions. Discharged, improved, September 1, 1903.

Note. While the diagnosis of manic-depressive insanity has been made in each of the above cases, it is made as a sort of last resort rather than because condition presented completely fits the diagnosis.

SIB GROUP NO. 47

A. Female, No. 13269, single. She has had three admissions to this hospital at the ages of 16, 24 and 37, each attack lasting about three months. Family history is said to be negative. Each attack commences with restlessness, sleeplessness and over-activity, becomes noisy, talkative, shows exceedingly good characteristic flight of ideas, poses and sings, emotional tone is happy. Recovery is sharp without any noticeable depression. Always well in the interim.



GROUP NO. 47

B. Male, No. 18365, single, 16 years on entrance July 30, 1908. A family history is now obtained. Father drinks considerably. Mother is distinctly nervous. Seven children, four of whom died before 18 months of age. This boy was sickly and distinctly feeble-minded as a result of epileptic attacks. However, between epileptic attacks he is mischievous, very talkative, euphoric, continually annoying others by his activity and interference. No delusions or hallucinations. It is stated that after each epileptic attack mental symptoms reach their height. He then acts like a typical excited manic and closely resembles his sister, except that he is feeble-minded and epileptic. Here, we have a case where manic-depressive temperament or constitution is united with epilepsy.

SIB GROUP NO. 48

A. Female, No. 14803, widow, 44 years on entrance September 14, 1908. Family History: Brother said to be insane. Commitment papers state that she thinks she has committed an unpardonable sin, contemplated suicide, very much depressed, retarded, apprehensive. In hospital, depressed, retarded, somewhat confused, believes she has committed an unpardonable sin, wishes to die, sits in one place, is with difficulty persuaded to eat. Died of chronic gastritis April 11, 1909.

B. Female, No. 8406, single, 60 years on entrance September 14, 1908. Was in this hospital previously from September, 1903, to March 23, 1904. History states that she has been in other hospitals for short attacks. Always cheerful, quiet and sociable. The first known attack was a depression. In 1903, the second attack, also depression. No hallucinations or delusions. Memory was good, orientation perfect, was depressed and retarded. Recovered. Present attack, well oriented, depressed, thinks she is about to die, everything is wrong with her, no hallucinations, grasp on surroundings is firm. She recovered from this attack, but was kept in the hospital because of her advanced years. January 25, 1915, she was placed in family care. Diagnosis, manic-depressive insanity.

Note. In this family the onset of the psychoses seems to have been around the involution period, but what may be called the manic-melancholic trend of the psychoses makes manic-depressive insanity the nearest Kraepelinian entity covering the condition.

SIB GROUP NO. 49

A. Female, No. 7857, married, 45 years on entrance February 28, 1879. Family history: Father was insane. On admission patient was much disturbed, refused food, was tube fed. Thought husband and friends were all dead, thought she had caused much of the trouble in the world, retarded, depressed. Recovered October 24, 1879.

B. Female, No. 8964, married, 44 years on entrance August 23, 1883. Had previous attacks. Suffered from melancholic attacks for some months. Thinks she is coming to want and has committed the unpardonable sin, sleepless, appetite poor, attempted suicide by hanging. In hospital, very much depressed, had to be urged to eat, thought things were continually going wrong. Improved very much and was discharged November 15, 1883.

SIB GROUP NO. 50

A. Male, No. 9795, married, 43 years on entrance May 12, 1866. Commitment papers stated, "Ill for a long time, now sleepless and excitable." In hospital, depressed, confused, has "foul disease," a bad odor issues from him, is suicidal, wrings hands, is agitated, cries "My God, My God," continually. Gradually improved and was discharged June 12, 1866.

B. Female, No. 9827, single, 23 years on entrance June 5, 1886. Excited, noisy, incoherent, prays continually, constantly excited and exalted, "entire incoherency in thought and action," tears off clothing, never rests day or night. Recovered entirely September, 1886.

In the following cases there is a distinct doubt as to the nature of the psychosis in both the sisters. They are placed in this group provisionally only.

SIB GROUP NO. 51

A. Female, No. 16387, married, 25 years on entrance May 20, 1905. The father was a heavy drinker. Mother's side was said to be negative. Early life: Average student, nervous. Attack of grip with mastoiditis. Became delirious, talkative, destructive, said people were cutting her open, no hallucinations, lost much weight. In the hospital was very flighty and active, answers were irrelevant and with evident intent to be humorous, was silly and evasive. The following is a copy of a letter written by the patient:

"Dear Sir: As I have received your letter last evening, I thought I would write and let you know that if you would not come after me soon there will be another house a fire. Monday I had a very blue day it makes me feel how notty you was to me sometimes, it drew the tears to my eyes. I help the girls in the dining room to wipe a few dishes and washed the floor and dusted a little. Not one kiss nor even the real molasses kisses. Tell Brother Billy write me a long & short letter. Will you please send me a pompadoir comb or bring me one in respects of Mr. Martin looking glass, or Arthur Wheaton ale mug name after the fly of the Jue's pants not sister Julia. Julia please get me a box of paper when you come for I will try and do as much for you some day, for your poor fat sister has been dreadful sick. Love to all the folks take care of Mary and Josie. Not the big Joe, of course or because you know what I mean please give a scratch on the palm of hand and I will make your ears ring, after the ring of the hole of the brown bread or the patient of key hole."

She was discharged December 23, 1905, said to be well. In the interim was said to be well.

Second admission, No. 19524, following a laparotomy for uterine trouble. Became excited and talkative, said she was sweet sixteen and had never been kissed. Answers were irrelevant. Memory said to be good, occasionally restless. In hospital, showed marked tendency to rhyme, the following being one of her answers: "Yes, I was here in the hall, ball, gall, mall." Question: In the which? Answer: "Witch hazel and Balm of Gilead." She did not recover from this attack. Disease became worse. Said she heard her husband loitering about the building. Believes when she was operated upon, they tried to bury her, and put her in a hearse, she had leprosy, was buried, and is now a spirit, says she hears her husband listening around the build-

ing, her husband had tuberculosis, has cold feet, apathy and delusions developed, has distinct delusions directed against husband, believes he tried to kill her, was frequently talkative and noisy, she refused to eat anything but bread and pudding, developed severe diarrhoea, and later lesions on the hands and face which were typical of pellagra. She became much worse and died February 2, 1917. Autopsy performed.

Diagnosis in this case is exceedingly difficult. The onset of the psychosis seems to have been of an acute confusional type, following somatic disorder. Second attack commenced in the same way, following a laparotomy, which became chronic and at all times presented a complex picture neither clearly dementia praecox nor manic-depressive insanity. To cap the climax she developed a pallagroid condition and died.

B. Female, No. 19197, single, 28 years on entrance May 5, 1910. Double ovariotomy several years ago, at the age of 16. At 25, became restless, complained of headaches, was nervous and irritable. On entrance to the hospital, at the age of 28, was quiet, dull-looking, said that voices tell her that she is guilty of murder, has committed unpardonable sins, something is going to be done to her, very markedly retarded, is with difficulty persuaded to eat. Recovered in three months.

Re-entered the hospital, No. 22503, July 9, 1915, age 33 years. Same complaint: She is going to die, the heart and intestines are decayed, people in automobiles go past her house, hiss at her and call her a murderer. At present in the hospital, is absolutely mute or else mutters and answers so that what is said cannot be made out. The expression of face is extremely apprehensive. Is slightly negativistic. Her mutism or retardation is so profound that it is impossible to make further mental examination.

Note. As in the sister's case, the difficulty arising in diagnosis is great. There are features in the case which resemble praecox, yet it seems to me the main change is in the mood, depression being the most profound symptom, and the delusions are entirely harmonious with them.

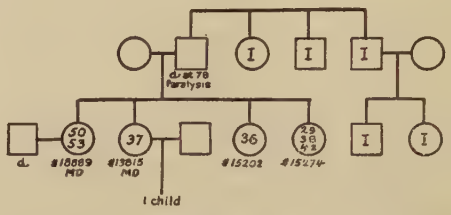
SIB GROUP NO. 52

A. Female, No. 15274, single, 29 years on entrance September 14, 1901. Family history: Father died at 78 of paralysis. Father's sister was insane, as were also his two brothers. A niece and nephew were insane. The mother's side is said to be normal. Early history, this girl was bright, but immoral. Onset of psychosis after father's death. Was noisy, talkative, flighty, profane and euphoric, showed psychomotor activity to a marked degree. Recovered. Had two other attacks October 4, 1910, to August 26, 1911, and August 30, 1915. Recovered perfectly from each attack. Was well in interims.

B. Female, No. 18889, widow, 50 years on entrance September 21, 1909. Markedly depressed, has occasional hallucinations of sight and hearing, says it always seems like night to her. Recovered, was discharged January 19, 1910.

April 24, 1912, re-entered the hospital. Depressed, melancholic, no insight, past sins worried her, has hypochondriacal ideas, mild delusions of persecution. Recovered and was discharged January 19, 1913.

C. Female, No. 15202, single, 36 years on entrance October 26, 1901. Patient had previous attack at 33. Present attack: Somatic ideas prominent, suicidal, says she never can sleep or work, no other delusions or hallucinations, retarded, memory good, oriented. Discharged, markedly improved, August 16, 1902.



GROUP NO. 52

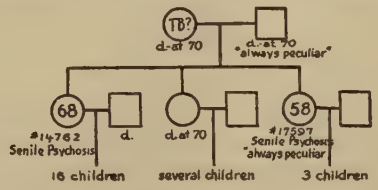
D. Female, No. 13815, single, 37 years on entrance June 1, 1898. Erotic, psychomotor activity, memory good, mild ideas of conspiracy. Recovered and was discharged September 5, 1898. Well in the interim.

Re-entered October 26, 1901. Became married, during pregnancy was depressed, suicidal, retarded. Remained so for six months and recovered. Had two other attacks in this institution from which she recovered, one for depression, the other excitement.

In this next group are cases in which senile psychoses and involution psychoses were present in members of the same generation.

SIB GROUP NO. 53

A. Female, No. 17597, married, 60 years on entrance December 26, 1900. Father had always been very peculiar. Died at the age of 70. Mother



GROUP NO. 53

died at 70. Patient is the youngest of three children. She had a fit after the birth of her second child. At 32, was irritable. After the birth of the third child unreasonable for a short time. At menopause was irritable, unreasonable, had hypochondriacal ideas and insomnia from which she recovered. At all

times an irritable, stubborn, negativistic person. In hospital, said she was very wicked, could not be forgiven, incoherent and irresponsible, at times untidy, scolds, talks constantly, says the medicine is poisoned, very negativistic and resistive, very profane. Died June 25, 1902, enteritis, inanition.

B. Female, No. 14762. It is now said mother died of tuberculosis, though this is doubtful. Was blind following grip, restless, became demented and helpless, untidy. Died in June 1902, of pneumonia.

Note. The first of these individuals was peculiar throughout life and final psychosis seems merely to have been the culmination of an abnormal personality.

SIB GROUP NO. 54

A. Female, No. 18534, married, 56 years on entrance January 9, 1909. Family history unknown. Had four children, three still alive. Delusions of reference and persecution of vague character, restless, threatening, memory good, apprehensive and hallucinated. Died of cardiac condition September 10, 1909.

B. Female, No. 20590, married, 65 years on entrance February 24, 1913. Had attempted suicide, was despondent, delusions of persecution of vague character, no hallucinations. Died of carcinoma.

SIB GROUP NO. 55

A. Female, No. 16877, married, 65 years on entrance February 21, 1907. For one year has had a loss of memory, disoriented, confused and feeble. Died of acute enteritis.

B. Female, No. 14732, 75 years on entrance May 16, 1905. Onset for two years. Condition as previous sister. Died December 15, 1909.

A. Female, No. 22684, single, 75 years on entrance September 7, 1916. Is feeble, defective memory, vague delusions of persecution against nurses, irrelevant, incoherent, querulous and untidy. Question of arteriosclerotic insanity or organic dementia.

SIB GROUP NO. 56

B. Female, No. 22562, widow, 60 years on entrance June 21, 1916. Has active auditory hallucinations, fairly oriented, defective memory, apathetic. It is said that the onset was at 45. Delusions of persecution at that time, ideas of poisoning prominent, delusions of reference. Dementia praecox is probably the correct diagnosis in this case.

A nephew of the two sisters is at present in the institution. Diagnosis, dementia praecox.

In the following three cases, alcoholic insanity occurred in members of the same family group.

SIB GROUP NO. 57

A. Female, No. 17937, married, 26 years on entrance September 9, 1907. Very clean-cut case of delirium tremens. Recovered quickly and was discharged October 5, 1907.

B. Female, No. 18089, single, 22 years on entrance June 7, 1908. Briefly an immoral, feeble-minded alcoholic who recovered quickly from the confusional state in which she entered.

SIB GROUP NO. 58

A. Female, No. 15019, married, 29 years on entrance June 11, 1901. On entrance saw rats, cats, dogs and mice, conversed in a rambling manner, became well oriented, but hallucinations of hearing and sight persisted for some time, had delusions of reference which persisted for a short time and then disappeared. Discharged recovered.

B. Female, No. 15990, married, 45 years on entrance June 7, 1903. Condition similar to sister's, except that there is a preliminary stage of visual hallucinations. Acute alcoholic hallucinosis, characteristic type.

SIB GROUP NO. 59

A. Female, No. 18853, married twice. Steady drinker for last 15 years. Short attack of confusional insanity from which she recovered completely.

B. Female, No. 18848, characteristic attack of delirium tremens. Recovered.

The following group of cases may be termed miscellaneous, in that somewhat different psychotic types were presented in the different siblings or else they are not easily classified.

SIB GROUP NO. 60

A. Female, No. 12890, married, 56 years on entrance February 26, 1896. Past history: No education; always cheerful. Present illness began on the December before admission. Was violent, afraid of being killed, did not sleep well, had pain in the head, attempted to jump out of the window twice. In hospital, was slightly excited, had active delusions of poisoning and persecution, auditory hallucinations, at times excited and noisy, conversation incoherent and irrelevant, active delusions, continued noisy and scolding at times. At present in hospital, demented, apathetic, still resistive and hallucinated. Though the hospital diagnosis is dementia praecox, an involution type of psychosis is perhaps more fitting as a diagnosis.

B. Male, No. 17862, widower, 65 years on entrance July 15, 1907. No previous history obtained. Said to be dull and excessively emotional at times for years. Memory impaired, tremor of fingers, hands, and facial muscles, conversation irrelevant, no insight, has visual hallucinations, delu-

sions of persecution, is irritable, restless and very excitable. Toward end of stay became somewhat apathetic. Died October 2, 1907, of acute enteritis. Diagnosis, senile dementia.

Note. There is no difference of any great importance in the two psychoses, except that the hospital incarceration occurred earlier in one case than in the other.

SIB GROUP NO. 61

A. Male, No. 13078, single, 26 years on entrance July 15, 1890. Patient is an epileptic with short periods of restless excitement following each attack. Between attacks, quiet, somewhat demented, no hallucinations or delusions.

B. Male, No. 18507, single, 35 years on entrance December 17, 1908. Has hallucinations of sight and hearing and feelings of influence, electricity is playing on him, he gets a message by means of a "catawitica," memory impaired, disoriented and demented. In hospital at present time. Diagnosis, dementia praecox.

Note. In these siblings, epilepsy with dementia praecox.

SIB GROUP NO. 62

A. Female, No. 13496, widow, 42 years on entrance June 23, 1897. Father died of heart disease, used alcohol to excess. Two sisters and one brother living. Following the death of a sister, patient became sleepless, depressed and agitated. Developed the delusion of electrical influence playing on her. Delusions of persecution directed against the neighbors. Very talkative at first, but not distractible. Repeats her sentences over and over again. She was discharged as improved in two months. Re-entered March 18, 1900, No. 14610. Was irrelevant, confused, negativistic, often quite excited, became violent and hallucinated, became demented, would not answer questions. Was discharged to Medfield March 18, 1906, where she is at present. Diagnosis here and there, dementia praecox.

B. Female, No. 20561, widow, 51 years on entrance February 15, 1913. Says a man came to her room at night and choked her. She makes signs with fingers suggesting secret orders. Her history is that she was married at 18, deserted; married again at 26; has two children, one miscarriage. For five years has been considered insane; that is, since the age of 46. Voices say that food is poisoned, restless, sleepless, thinks her son is going to be put in an electric chair. In hospital, conversation is rambling and incoherent, says she feels sharp nails in the bed, somebody is choking her by a secret influence, is disoriented, confused, excited, memory impaired, hallucinations of hearing. No change during her stay, at times apathetic and listless, often crying, agitated and showing her restlessness. Died of cerebral hemorrhage January 28, 1915.

Note. It may be stated that the two psychoses resemble each other very closely, having an onset about the same time in life with delusions of per-

secution, hallucinations of hearing, restlessness, agitation, and finally dementia.

Had the psychosis occurred in either of the two cases at 25, no one would have doubted the diagnosis of dementia praecox. It is one of the fallacies of modern psychiatric practice that the age of onset is given too great an importance. It is true that the same general psychotic type may have its onset at puberty, involution and senile periods and differs somewhat at each period, but the difference is not so great as, for example, occurs in lobar pneumonia at corresponding periods of life. There is a certain coloring which each period of life gives which ought not to distract one's attention from the fundamental features of the psychoses. In these two cases, I think the diagnosis of dementia praecox can certainly be made despite the fact that the first symptoms occurred at the involution period.

SIB GROUP NO. 63

A. Female, No. 16598, 62 years on entrance October 15, 1904. Onset was at 30 with delusions of persecution, hallucinations of sight and hearing, apathetic, occasional catatonic attitudes and mannerisms. In hospital, speech was of a salad type and a letter she wrote to a relative illustrates the same phenomenon.

B. Female, No. 22543, widow, 86 years on entrance January 11, 1916. The onset was at the menopause. Had always been peculiar. Active symptoms developed only lately with delusions of persecution, especially in regard to property. Violent at times, defective memory, confused and irritable In hospital at present, sits around on bench, is apathetic except when disturbed, when she becomes exceedingly irritable and scolds away. Probably hallucinated, dementia profound, delusions of persecution concerning nurses.

The family history in this case is interesting. (See chart.) Maternal grandmother was insane at one time. The mother was insane for a short period at the menopause, was always peculiar, and hard to get along with. The mother's sister, an aunt, was insane "on religion." The father was normal. Of the four children born of this union, the two boys died early in life. Cause of death cannot be ascertained. The two sisters were insane; one unquestionably dementia praecox, the other developing a psychosis late in life on a fundamentally abnormal personality. Has a hospital diagnosis of senile dementia.

I see no fundamental difference in the psychoses presented by the two sisters except that one was slow in development with the main focus of incidence of disease late in life, whereas the other had the focus of incidence of symptoms earlier in life.

SIB GROUP NO. 64

A. Male, No. 18394, married, 52 years on entrance, October 27, 1908. One brother committed suicide. Patient had been worried over business troubles for some time. Became noisy, restless and depressed, had to be fed. In

the hospital, negativistic, depressed and confused. Recovered quickly. While in hospital, had several short attacks of similar condition. During interim was entirely well. No hallucinations noted and no definite delusions. Left August 5, 1910, and is said to have been well since. Differential diagnosis in this case between catatonic episode and depression. The negativism and confusion incline one towards catatonia, but there is nothing in the case that definitely contradicts a diagnosis of manic-depressive insanity.

B. Male, No. 18517, single, 56 years on entrance December 26, 1908. Was always considered feeble-minded and irritable. Very eccentric, wearing an overcoat in August. Became deaf and rather helpless and was sent to this hospital. The hospital history shows nothing but very distinct feeble-mindedness, eccentricities and deafness. No hallucinations or delusions noted.

Note. One sibling had a psychosis which may be either catatonic or manic-depressive; the other sibling showed a distinct feeble-mindedness with, however, eccentricity in personality.

SIB GROUP NO. 65

A. Female, No. 18854, single, 44 years on entrance August 18, 1909. Past history: She had one illegitimate child. Became deaf 12 years before admission, since which time has always heard voices in her head. Consumes a large quantity of Peruna (a patent medicine which at that time contained a good deal of alcohol).

Present Illness: Brooded over the illegitimate child, very depressed, suicidal ideas prominent. On account of this was sent to the hospital. In hospital, said to have auditory hallucinations, reproaching her for the illetigimacy of her child, very depressed, discouraged, suicidal, memory was good, orientation perfect, grasp on surroundings firm. Made quick recovery and was discharged September 28, 1909. Denied that she had been hallucinated at that time.

Note. The psychosis seems to have been a short depression which may be classed in the large group of manic-melancholia.

B. Female, No. 18855, married, 48 years on entrance August 18, 1909, same day as sister. Past history: Hysterectomy four or five years after marriage, at the age of 28 years. Was in the Rhode Island State Hospital at 29 for one year.

Present Illness: Began to have epileptic convulsions when quite young. Typical grand mal attacks, periodic. Memory defect became prominent early. She would have periodic attacks daily for about a week, would then show impaired memory, be cross and irritable, then recover, be fairly normal for about two weeks, and then a further series of attacks would occur. This cycle has been prominent for the last three or four years. Hospital history: Orientation good, memory defective, occasional hallucinations of sight, cheerful, quiet, neat and tractable. Discharged on trial October 30, 1909. Diagnosis, epileptic insanity.

SIB GROUP NO. 66

A. Male, No. 12252, married, 39 years on entrance July 2, 1894. Had a previous attack, when he refused food. Says he will cause trouble between the two churches, the Protestant and Catholic. Prays loudly and violently in public places, very noisy and violent at times, at other somolent. This attack commenced with insomnia and restlessness. In hospital confused, disconnected conversation, indecision prominent, said he did wrong by being married by a justice of the peace, his people are ashamed of him because he criticizes the churches. As he converses, he fixes his eyes upon ceiling and exultantly exclaims, "The ceiling looks bright—everything looks bright if I follow the light." Is restless, depressed, but depression relates to his delusions and hallucinations, is markedly agitated and weeps. Very symbolic: says, "They look like stars in heaven, they mean right or wrong. One tries to coax, the other to threaten. One is the Free Masons, the other the Catholic Church." On one occasion struck attendant and bit him on arm. Mood very fluctuant, at times distinctly elated, unreliable and restless. Recovered October 25, 1894.

B. Male, No. 19235, 53 years on entrance June 3, 1910. One sister insane for 10 months. One brother died of typhoid, one sister died of croup, one died of abscess. In temperament patient was always worrisome. Had a bad heart and much economic and domestic trouble, became apprehensive and worrisome, had auditory hallucinations of threatening character, became very much confused and attempted suicide, refused to eat, was tube fed. Died of gastrointestinal disorder June 28, 1910.

Note. The second case may be classified as an involution melancholia because of the marked agitation, the apprehensiveness, the refusal to eat, and short, fatal course. The brother's psychosis is more difficult to place. There are two distinct attacks separated by 10 years, both attacks being short, characterized on the whole by depression, agitation, the feeling of being the center of a conflict, marked symbolism and recovery. A sister also had a short psychosis having the same general features. Very likely all the patients had the depression of manic-depressive insanity.

SIB GROUP NO. 67

A. Female, No. 13597, single, 22 years on entrance November 3, 1897. The commitment paper says, "She puts food in the fire, runs away, assaults her mother." In hospital, very violent and destructive, masturbates openly and excessively, difficult to know whether she is reacting to hallucinations, talks rapidly but coherently. Died of exhaustion November 20, 1897; that is, 17 days after admission.

B. Male, No. 18728, married, 45 years on entrance June 12, 1909. Acute alcoholic hallucinosis. After an alcoholic debauch developed the delusion of persecution, that he was followed by detectives because of being blamed for thefts and murders. He heard voices reproaching him, became sleepless, emotional suspicious of his food. Started to recover shortly after admission, recovered completely in seven months. Was discharged May 7, 1910. Diagnosis of attending physicians was acute alcoholic hallucinosis.

SIB GROUP NO. 68

A. Male, No. 16211, 43 years on entrance December 14, 1907. On admission, had a temperature of 100, pulse 74, respirations 22. Was very active, shouting "Glory to God," inaccessible, hallucinated. This changed to a muttering delirium. Lobar pneumonia was diagnosed and he died of the same a week after admission.

B. Male, No. 16201, 38 years on entrance December 5, 1907. History is scanty. He had been in an asylum in England two years before with a short attack. He had exactly the same symptoms as brother—was excited, restless, became delirious, had a high temperature, and lobar pneumonia was diagnosed.

Note. In these two brothers, a remarkable coincidence brought them to the hospital within a week of each other with exactly the same trend of symptoms; that is, signs of lobar pneumonia, a short, excited period, and then delirium and death. In the one brother, it is stated that he had had a previous attack two years before.

SIB GROUP NO. 69

A. Male, No. 13630, married, 42 years on entrance November 23, 1897. Past history: He has always been melancholic in disposition; used alcohol and tobacco, but only occasionally. Present illness began November 16, 1897. He thought people were following him about and talking about him, he became sleepless, had nightmares and pain in the head, became restless and threatening. Physical examination showed lively knee-jerks, tremulous hands, rather unsteady gait and station, speech thick. Hospital history: Coherent, quiet and tractable, memory good, auditory hallucinations, voices talk to him all the time, swearing and cursing. General condition improved, continued to hear voices, but he realized they were imaginary. A complete history obtained later showed that he had been drinking heavily for some time before entrance and that he was a heavy drinker rather than a moderate drinker. The diagnosis was toxic insanity. (Acute alcoholic hallucinosis.)

B. Male, No. 13781, 39 years on entrance May 6, 1898. Past history: Always cheerful, moderate user of liquor and tobacco. Became low-spirited because he was out of work. Had had a previous attack at the age of 15. Was sick seven weeks and fully recovered. Present illness began six months before admission. Came home from work tearing clothes, talked constantly about not being able to work, was troublesome to the neighbors. In hospital, a systolic murmur transmitted to the axilla was found. After a very short period he had delusions of persecution, said people in the house thought gold treasure was buried there, concealed in the cellar, and wanted him to get out so they could get it. Electricity was worked into his head and caused it to buzz. His condition quickly changed for the better, delusions disappeared as did hallucinations. He was discharged June 1, 1898, much improved. It is possible here also that alcohol was used to excess. (There

is a statement made that he had a similar attack at the age of 16, when alcohol as a cause seems unlikely.) If alcoholism in his case is admitted, then the psychoses are very similar.

SIB GROUP NO. 70

A. Female, No. 7937, single, 22 years on entrance August 25, 1879. Unruly, destructive, violent, noisy. Always considered feeble-minded. In hospital, at first tractable, industrious and mild, jealous of other patients if nurses attended them, demonstrative toward nurses, later called herself by some other name and would not answer to her own name, irritable and quarrelsome, thinking that others were continually trying to annoy her. She was discharged August 8, 1880. Diagnosis at that time, feeble-mindedness plus a possible psychosis.

B. Female, No. 15313, single, 50 years on entrance January 21, 1902. One sister half-witted, No. 7937. Patient was considered dull as a child, got only enough education to read and write, was always nervous, excitable and easily frightened. Present illness began in September, 1901. Mind found to be weakened during the course of typhoid fever. Neuritis gradually developed. Delirious during the fever. As fever left, appeared to collapse. Visual hallucinations developed; paralysis in extremities appeared. Physical examination showed absent knee-jerks, foot drops on both sides. Forearms held in flexed position with atrophy of muscles of arms and legs. She was confused and hallucinated. She died suddenly April 1, 1902, when some sort of pelvic tumor was discovered.

SIB GROUP NO. 71

The family history, which was very carefully investigated, is negative so far as parents, grandparents and collateral relatives are concerned. This family history was obtained by field workers, correspondence and much careful effort. The father is alive at the age of 56, is not well because of severe attacks of asthma. Personal physical examination made of this parent reveals the fact that the knee-jerks are absent and he is somewhat unsteady in his gait. Pupils are somewhat dissimilar in size and react poorly. Wasserman taken of the blood was negative. No opportunity for a repetition was given. Mother died at the age of 54 of Bright's disease. (The family chart is appended. It will be seen that of 10 children who are known, four presented a condition similar to this one.)

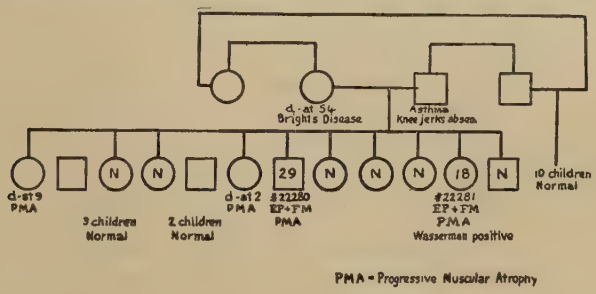
A. Male, No. 22880, single, 29 years on entrance December 14, 1915. Is almost a complete idiot, has had epileptic seizures two or three times per month for many years. The physical examination shows main en griffe on the left side, and incomplete but similar condition on the right. There is marked atrophy of the muscles of the arms, especially the supraspinati and triceps. Very marked lordosis. There is complete foot drop with atrophy of the muscles of the tibialis anticus on both sides. There is a resulting clubfoot contracture. Pupils are equal, regular; react well to light and

distance. Abdominal skin reflex and cremasteric very sluggish. Knee jerks absent, plantars absent.

For a short time attempted to walk and could talk a little at the age of seven, but gradually he has lost the ability to talk so that now he can say nothing and for years has been unable to walk, becoming more and more helpless all the time.

It is stated by the parent that this condition developed gradually and has been growing worse until the care of this patient and his sister, whose history follows, became impossible at home. He died following a general infection, apparently resulting from a skin wound.

B. Female, No. 22281, single, 18 years on entrance December 14, 1915. At present in hospital. History was exactly that of her brother, except that she learned to walk and talk somewhat better than he did. Epileptic attacks were more marked in her case.



GROUP NO. 71

It is now learned that the father's youngest brother married a sister of his wife so that the same stock entered into this brother's children as did into the family under consideration. Of the 10 children resulting from this union, all lived, none were feeble-minded, insane or defective in any way. Several are married and have children who show no similar defect.

Physical examination of this patient showed practically the same condition as did the brother. There is, however, in this case positive Babinski on the right side, a positive Gordon on this side. Left side shows neither Gordon nor Babkinski. Oppenheim is negative on both sides, knee-jerks and ankle-jerks missing. On January 10, 1916, blood serum gave positive reaction to the Wassermann test for syphilis. (Test performed in State Laboratory, Boston.) February 3, 1916, blood serum gave a negative reaction to the Wassermann test for syphilis; and February 17, a doubtful reaction to the Wassermann test for syphilis—all tests being performed in the same place. January 21, 1916, spinal fluid entirely negative to albumen, globulin, cells and Wassermann. A more detailed examination of this patient was made. The supra- and infraspinati are decidedly atrophied. Small muscles of hand, especially interossei and muscles of hypothenar eminence are atrophied. Hand is held in a constant claw position with ulnar devia-

tion with some ankylosis at the wrist joint. The feet are in a marked position of foot drop also pes talipes. The small toes, and to a lesser degree the large toes, are sharply flexed downward. There is atrophy of the extensor group of muscles and the tibialis anticus. Facial muscles apparently intact. Bulbar muscles intact. Skin and tendon reflexes everywhere absent. Patient cannot talk and seemingly understands nothing.

On March 2, 1916, spinal fluid repeated, negative reaction to the Wassermann test for syphilis. Blood serum at this time gives doubtful reaction to the Wassermann test for syphilis.

Note. The interesting thing in this group lies in the fact that what seems to be a familial, progressive, muscular atrophy is found in four members out of nine. Two have been closely observed, one being in the hospital at the present time. All the relatives, except in the immediate family, are healthy. *A rather interesting biological experiment was carried out when the brother of the father and a sister of the mother married and had 10 healthy children. This would seem to rule out any distinctly hereditary factor in the sense of a disturbance of gametes or anything of that kind. Some disease process has crept in, damaging the progeny.* This is borne out by the physical examination of the father, and syphilis is strongly suspected in his case. One Wassermann test on one of the two children in the hospital (the only one examined in this way) was positive, but several repetitions have been either negative or doubtful, the spinal fluid being doubtful. Nothing further can be stated concerning this case, except that idiocy and familial progressive muscular atrophy are rarely found together. In this case there is undoubtedly organic brain disease as well as organic cord disease.

The following three cases are given in some detail because conditions presented are interesting, details are abundant, and they have a special interest aside from the diagnosis in each individual case:

SIB GROUP NO. 72

A. Female. No. 8554, married, 40 years on entrance May 9, 1882. Family history not obtained at this time. "Patient lost three children within 10 days and has been breaking down in mind ever since." Incoherent, believes that her children appear to her and talk with her, excited at times, generally depressed, weeps a great deal. In hospital, usually very mild and quiet, quite apprehensive, fears harm to self, fears she is to be cut up by surgeons, much depressed, inclined to lie about on the sofa without working. Gradually improved, became more cheerful, cried less, worked more, and was discharged February 18, 1885.

A recent history which has been obtained states that she has been quite well most of the time since. Carried on her household work, always rather nervous and easily depressed, but no active mental symptoms, no dementia.

B. Female, No. 14199, married, 37 years on entrance June 14, 1899. In disposition, always nervous, easily frightened. Ordinary mental ability.

Three months before she was sick, experienced a burning sensation in side and flowed profusely. Attended by physician and sent to hospital where she had curettage. Flowed for three months continuously. After operation, began to talk queerly, saying that she was dying, cried nearly all the time, lost interest in and all love for children and home, became untidy, heard people talking about her, sat around without working, complained of severe headaches, threatened suicide, and on several occasions made desperate efforts to end her life.

Physical examination in hospital negative. Mental examination showed depression, restlessness, agitation, no memory defect. Talked about recent operation continuously. No hallucinations of sight or hearing. Shortly after entrance started to improve and on June 16, was discharged, recovered.

Re-entered the hospital November 2, 1909, age 52 years. Had been well in interim. Two months before admission, a fly flew into her ear. A doctor syringed out the ear, informant thought he syringed it too long. Began to show signs of mental trouble. She became nervous, restless, would not take nourishment, had spells of laughing and crying, became markedly apprehensive, was afraid to eat, generally much depressed. Physical examination negative. On entrance, memory good, no hallucinations noted, restless, excitable and emotionally agitated, cries a good deal, reveals no definite delusions. As before, improved and was discharged April 20, 1910, recovered.

C. Female, No. 23141, married, 62 years on entrance July 12, 1917. Family history obtained at this time shows the following: Father and mother said to be well. A maternal first and second cousin in this institution. Of the brothers and sisters of this patient, two sisters were in the hospital, a brother died of tuberculosis at the age of 14, one sister is alive at 70. Patient suffered with severe vomiting during each of her 10 pregnancies. A year ago she had an attack of grip, complained of pain in the left shoulder, which was diagnosed by some neurologists as neuritis. Following this, she began to lose weight gradually, appetite became poor, has lately become restless, talkative and complaining, threatening at times, but generally very much depressed. Talked suicide. Because of this, she was committed to the hospital.

Physical examination showed a rather worn-down old woman.

Mental examination shows simply an anxious, restless woman, who is well oriented as to time, place and person, has good memory for recent and remote events, no hallucinations, no definite delusions elicited, flow of thought somewhat irrelevant and incoherent, at times she appears slightly confused. On the whole pleasant and tractable.

Note. The three individuals have practically the same history. Following some disturbance—as in the first patient, the loss of three children; in the second and third, sickness—there follows a disturbed, apprehensive agitated mental state which is very troublesome to those around them, but which passes quickly away. In a sense, it is a symptomatic psychosis. It resembles the emotional reaction of normal individuals to similar circumstances, except that it is exaggerated and prolonged. The reaction will be discussed further in this paper. The condition does not belong to demen-

tia praecox and perhaps may be called agitated depression of manic-depressive insanity. The two cousins, who are uncle and niece, were both dementia praecox.

SIB GROUP NO. 73

A. Male, No. 8052, married, 36 years on entrance April 15, 1880. One maternal uncle, No. 1199, in this institution. Diagnosis, acute melancholia. Patient's early life is said to have been normal, always easily depressed and excited. Onset of this psychosis sudden. Threatened to kill his wife, was noisy, running wildly about the streets. Was finally secured, almost naked. Confused, incoherent, noisy and destructive. This lasted for three months when he gradually quieted down into a stupid, apparently demented, state, seldom speaking and avoiding interviews, rather resistive. No hallucinations or delusions elicited. He slowly emerged from this and during the last two months of his stay improved to the point of recovery.

Re-admitted, No. 17017, 63 years on entrance September 13, 1915. Was well in interum except a year before entrance when he left his home with $200, spent his money freely, went to New York and walked the whole length of Long Island. Was very active, felt too buoyant, felt himself exalted above other men. This attack commenced suddenly. He threatened to kill several persons, thought he must kill seven men before he died, attempted a criminal assault on his daughter just before entrance. Excitement lasted only a few days. Thereafter, he was slightly exhilarated, talked freely, but showed no undue verbosity. Orientation good, memory good.

February 14, he says he has a new tooth coming at his age of life. Says, "I can go to bed and wake up any time I want to. At one time I needed a dollar and I wanted to wake at a certain hour. I was very sleepy, but suddenly I heard a rap. That showed me what the Almighty could do. Those raps were a warning. I buried my wife a short time after that." He was fault-finding, somewhat irritable, but gave expression to no delusions or hallucinations. Showed no dementia. February 1, 1907, was discharged to the State Board of Insanity, condition not improved. He seems to have settled down from an exhilarated state to a seclusive, rather aggressive, suspicious mental condition without noticeable dementia or distinct hallucinations or delusions, although both these were suspected.

B. Male, No. 8186, married, 28 years on entrance November 30, 1880. Eight years ago after sunstroke was insane. Was violent and dangerous, showed religious excitement, temperate, became excited, dictatorial, said his wife was underground, and that she was dead, ordered people out of the house under threat to kill, walked about the streets wildly, knelt in the mud and prayed. Obstinate and resistive on entrance.

In hospital, quickly became quiet and at time of discharge, August 31, 1885, showed no active symptoms.

C. Female, No. 13626, widowed, 47 years on entrance, November 22, 1897. A family history was now obtained. The father died of cancer of the stomach. Mother died of old age. This patient had five brothers and sisters.

One is insane and blind at home, one committed suicide. Two brothers considered above. One sister, patient in this institution. It is stated that father was insane at times for short periods.

Patient always melancholic in disposition. Showed signs of mental change at death of her first husband, 16½ years ago. At that time had visions and threatened to drown herself. She became hyperreligious, excited, took no care of her home.

In hospital, says she became converted when in her teens. Was overwhelmed with the sense of sin when suddenly the room was filled with a light, visible and yet invisible, and she felt the Peace of God fill her heart. Since then she has frequently heard the voices of relatives who are dead talking to her and telling her what to do. She has had warnings, these voices telling her not to do certain things. In hospital, showed evidence of hallucinations and delusions of a religious character. She recently complained that the night watchman injured her by administering certain gases and that these produced a peculiar effect on her. Incoherent and irrelevant in answers. Throughout her stay she showed talkativeness, restlessness, apparent hallucinations of sight and hearing, mild delusions of persecution, and delusions of an exalted, religious nature. At times showed marked flight of ideas. She was discharged August 17, 1904, as a boarding-out patient, there being no particular change in her condition.

She was returned from boarding out; and shortly after re-entrance had a cerebral hemorrhage involving the right side. Died December 3, 1908, having been in a paralyzed state from the time of the first hemorrhage.

D. Female, No. 8075, married, 23 years on entrance May 17, 1880. "Usually mild in character, but violent if opposed. Suicidal, has beat her head against the wall, wants to go to the water. Said to have times of excitement, when she is dangerous to those about her." Excitement shortly passed away and she became dull, listless and somewhat depressed. Condition began to improve shortly after that and she was discharged as recovered October 11, 1880.

Note. The first of these patients, after an attack which seems to have been manic, settled down to a chronic condition which in certain respects resembles dementia praecox. The second had an acute excitement which may have been either catatonic or excited phase of manic-depressive. The third patient was diagnosed as dementia praecox by the hospital staff and distinct paranoid ideas with hallucinations and delusions, the chronic condition in general seems to favor that diagnosis despite the flight of ideas that occurred at times. Therefore, there is to be considered here the occurrence of manic-depressive and dementia praecox in the same sibling group. I myself am not satisfied with either the data presented or the place in the classification to which this patient belonged.

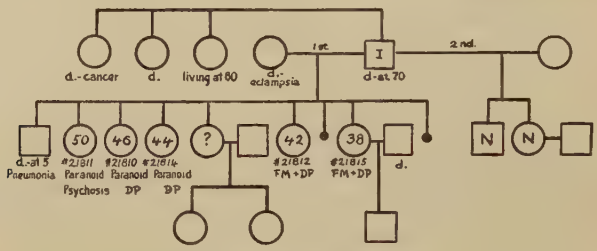
SIB GROUP NO. 74

This family, which has been described at length by Dr. John F. O'Brien in papers read before the Boston Society of Psychiatry and Neurology and

the New England Society of Psychiatry is here given because it shows a sort of transition from one type of psychosis to another.

Family history is here given for all the members of the family. Mother died of eclampsia following childbirth. Father died at the age of 70. For the last two years of his life he was "insane" and shared the delusions of his daughters. There were born to this couple nine children. A boy died of pneumonia at the age of five. The next two were dead when born, as a result of difficult labors, both being boys. Of the next six, all females, five were brought to this institution. The sixth daughter was interviewed, was suspicious, uncommunicative, and refused to commit herself concerning any beliefs she shared with her incarcerated sisters. Following Dr. O'Brien's presentation, it may be stated that there is a transition of symptoms from the oldest to the youngest.

A. Female, No. 21811, single, 50 years on entrance January 11, 1915. Early life was uneventful. Received a good education and was bright in school.



GROUP No. 74

Following her mother's death the father re-married. All of the daughters were bitterly opposed to the stepmother and made life very unpleasant for her. Some property was left them, and around this property the main delusions center. They believed that a gang of conspirators, including the prominent men of the city, the Catholic Church (they are Catholics), lawyers and doctors, town officials, the police department, etc., have attempted to make the property undesirable by persecuting the owners of it, these sisters. The means they have used have been noises, gases, animals of all kinds, electrical influences, sexual attempts, etc., all with the idea of making life so unpleasant that the sisters would sell the property.

This patient is oriented, coherent, generally pleasant, a good worker, no dementia. Her delusions are systematized and hallucinations have been either very fleeting or not at all prominent. Bodily health good. Emotional tone pleasant.

B. No. 21811, single, 46 years on entrance January 11, 1915. Patient shares the same delusions as the sister. They are not quite so coherently expressed, a little less systematized. Hallucinations of hearing and of smell have been rather more prominent in her case. She is not such a good worker, but is not demented. Fairly pleasant.

C. No. 21814, single, 44 years on entrance January 18, 1915. Patient is more poorly built than her sisters, not so bright, delusions are wide-spread,

incoherent, hallucinations of sight, hearing, and smell of the most fantastic kind are very prominent. She is of a very ugly temper, and when out on a short visit she shot and killed the police officer who was attempting to bring her back. She will not work, is resistive, very evidently is of lesser mental caliber than her sisters and is probably showing beginning dementia.

D. Female, No. 21812, single, 42 years on entrance. Patient is much demented. No hallucinations or delusions can be elicited. She is apathetic, indifferent, talks in a whisper, never been outside of her own home, has not the slightest memory of any educational facts, disoriented as to environment. The history is that she has been feeble-minded from the start. From her 22nd year she has rapidly deteriorated and become more and more seclusive.

E. Female, No. 21815, widow, 38 years on entrance. The patient has always been considered somewhat feeble-minded. Shares the delusions of her sisters, but expresses them in an extremely incoherent manner, is very quiet, retarded, shows either dementia or past feeble-mindedness, eats poorly. Has the habit of placing papers, handkerchiefs and various other articles in the front of her waist, is untidy and very seclusive. During her stay in the hospital she has deteriorated rapidly. At present is demented.

Note. These five sisters show a transition in psychotic type. The first patient may be called a paranoia vera or paraphrenia systematica. The second patient is possibly the same. The entrance of hallucinations of fantastic character would make most clinicians feel that paranoid dementia praecox was to be considered. The third patient is undoubtedly a paranoid dementia praecox. The fourth and fifth are dementia praecox, dementing, with delusions and hallucinations either disappeared or rapidly disappearing in the breaking up of the mentality. It is interesting to note that the father was insane in the last two years of his life, having a somewhat similar psychosis to his daughters. There is, therefore, in the second generation anticipation, increase in mental symptoms, with what appears to be a transition in the type of psychosis presented from one sibling to another.

In the previous paper it was stated that paranoid diseases in an ancestor stand related to paranoid dementia praecox and to dementia praecox in the descendant. It may also be true that true paranoia stands biologically related to paranoid dementia praecox in the same generation.

BRIEF ANALYSIS OF ABOVE CASES

As has been stated previously, statistics that relate to only a small number of cases have only a limited value and it is carrying a study to a ridiculous fineness when one attempts to make percentages of such statistics. All one can do is to discover trends and point out directions. The following table indicates first, the sexes of the

FAMILY NUMBER	SEX	OCCURRENCE OF FEEBLEMINDEDNESS	MENTAL DISEASES IN ANCESTOR
Those in which dementia praecox is diagnosed in all members of the fraternity in the hospital			
1	2 sisters	1	Gross defect in mother
2	2 sisters		Paternal side
3	1 sister, 1 brother	1	
4	1 sister, 1 brother	1	
5	2 sisters	2	
6	1 sister, 1 brother		
7	1 sister, 1 brother		
8	1 sister, 1 brother		
9	1 sister, 1 brother	2	Maternal side
10	2 brothers		
11	1 sister, 1 brother		
12	2 brothers	1	Maternal side
13	1 sister, 1 brother	1	
14	1 sister, 1 brother		
15	2 sisters	2	
16	2 brothers		
17	1 sister, 2 brothers	1	Maternal and paternal side
18	2 sisters		
19	2 brothers		Maternal side
20	2 sisters		
21	2 brothers		Paternal side
22	1 sister, 1 brother		
23	1 sister, 2 brothers	1	
24	2 sisters, 1 brother		
25	1 sister, 1 brother		
26	1 sister, 2 brothers	3	Maternal side
27	1 sister, 1 brother		
28	1 sister, 1 brother		Paternal side
29	2 sisters		Paternal side
30	1 sister, 1 brother	1	Maternal side
31	2 sisters		
32	1 sister, 1 brother	2	
33	1 sister, 2 brothers	3	Paternal side
34	2 brothers	1	Paternal and maternal side
35	1 sister, 2 brothers	2	Maternal side
36	2 sisters, 3 brothers		Paternal (?) and maternal (?) side
37	3 sisters, 1 brother		
38	2 sisters		

FAMILY NUMBER	SEX	OCCURRENCE OF FEEBLEMINDEDNESS	MENTAL DISEASES IN ANCESTOR
	Those in which dementia praecox is diagnosed in all members of the fraternity in the hospital—*Continued*		
39	1 sister, 2 brothers		Maternal side
40	1 sister, 1 brother		
41	2 sisters, 1 brother		
42	2 sisters, 1 brother		
43	1 sister, 1 brother	2	
44	2 sisters		Paternal side
45	2 brothers	1	
	Those in which manic-depressive insanity is diagnosed in all members of the fraternity		
46	2 sisters		
47	1 sister, 1 brother	1*	
48	2 sisters		
49	2 sisters		Paternal side
50	1 sister, 1 brother		
51	2 sisters		
52	4 sisters		Paternal side
	Those in which senile dementia is diagnosed in all members of the fraternity		
53	2 sisters		
54	2 sisters		
55	2 sisters		
56	2 sisters		
	Those in which alcoholic insanity is diagnosed in all members of the fraternity		
57	2 sisters	1	
58	2 sisters		
59	2 sisters		
	Miscellaneous cases		
60	1 sister, 1 brother		
61	2 brothers	1*	
62	2 sisters		
63	2 sisters		Maternal side

GROUP NO. 1—*Continued*

FAMILY NUMBER	SEX	OCCURRENCE OF FEEBLEMINDEDNESS	MENTAL DISEASES IN ANCESTOR
	Miscellaneous cases—*Continued*		
64	2 sisters	1	
65	2 sisters	1*	
66	1 sister, 1 brother		
67	1 sister, 1 brother		
68	2 brothers		
69	2 brothers		
70	2 sisters	2	
71	1 sister, 1 brother	2	
	Special cases		
72	3 sisters		Maternal side
73	2 sisters, 2 brothers	1	Maternal side
74	5 sisters	2	Paternal side

* Epileptic.

siblings involved; second, the incidence of "feeble-mindedness"[1] in conjunction with the other psychoses, that is, feeble-mindedness occurring together with some other mental condition in the patients presented; and, third, the incidence of insanity in the ancestors. It is to be emphasized that a positive history of "insanity" occurring in an ancestor is to be credited as true though its value is little without details of type. A negative history, a denial, or a claim of non-existence of insanity means nothing. In this respect the history of "insanity" in a family may be classed with the Wassermann reaction. When positive, this means, in the overwhelming majority of cases, syphilis. When negative, it does not exclude syphilis.

Group No. 1. As evident from above, the males and females are about equally represented. "Feeble-mindedness"[2] occurred 18

[1] I use the term feeble-mindedness here under protest. On history one has to depend on uncertain and poor observation. I make no great claim as to the value of these statements.

[2] The term feeble-mindedness is here used as given in histories, not because we are dealing with any unitary condition.

times in one or both members of the groups presented. That is to say, prior to the development of the psychosis, feeble-mindedness was a condition noteworthy enough to be given in a routine history taken at the hospital. Undoubtedly higher grades of feeble-mindedness, what is nowadays called the moron type, occurred frequently. At any rate, it is worth noting that in cases where dementia praecox occurs in several members of one generation, feeble-mindedness as a part of the development of the psychosis or as a coincidence is common. This is a decided contradiction to the opinion expressed by Singer in a later paper in which he emphasizes his belief that the two conditions mentioned rarely coincide. It is in accord with the opinion expressed previously, in which it is believed that dementia praecox in an ancestor tends towards mental deficiency in a descendant.

Group No. 2. The preponderance of sex was very greatly female. With so small a group of cases, however, no great stress should be laid on this fact. Feeble-mindedness occurred only once amongst these manics and then in the case of a feeble-minded epileptic boy. It would seem from the literature as well as from these cases, that feeble-mindedness occurs rarely coincidental with manic-depressive insanity, whereas it occurs commonly with family dementia praecox.

Group No. 3. In the four families here adduced, the sex was altogether female. No feeble-mindedness was given in the history and family mental disease was not manifested.

Group No. 4. Alcoholism. Three families, all females, one feeble-minded. History of family mental disease is not conspicuous.

Group No. 5. Miscellaneous group. Twelve families, the preponderance of sex slightly in favor of females. Epilepsy occurred twice in conjunction with the psychoses. Feeble-mindedness occurred three times in conjunction with the psychoses. The occurrence of family mental disease is given only in one case, which of course, is obviously a gross under-statement.

Group No. 6. Special cases. Three families, more females. Feeble-mindedness occurred twice in one family (dementia praecox as well). Family mental disease in all three families which happen to have been closely studied.

If now we discuss the definite question, do the psychoses of brothers and sisters tend to be alike?, we find that by far the greater number of the authors and the cases here cited are in agreement. The general conclusion may be stated as follows: In by far the great

majority of cases, the psychoses in brothers and sisters tend to be the same. So far as I know, only one author, Sioli, is in disagreement with this statement. All the authors, who have been cited at more length in the previous chapters agree, though the percentages of similarity in siblings differ.

Of the Taunton cases, the psychoses are essentially similar in all except the following cases: In case No. 11, in which one of the patients had general paresis. This cannot be cited as a dissimilar psychosis because general paresis and the ordinary organic brain diseases, such as tumor of the brain, cerebral hemorrhage, etc., are biologically very distinct from the psychoses that we have been considering. I have several cases in which dementia praecox or other "endogenous" disease occurs with general paresis. Case No. 25, here possibly true paranoia occurs with dementia praecox. Case No. 74 seems to show a direct transition from paranoia vera to paranoid dementia praecox, dementia praecox and feeble-mindedness. Cases Nos. 53, 54, and 56 are doubtful. In Case No. 61, epilepsy occurs together with dementia praecox. No. 64 is doubtful. In No. 65, one of the siblings had epilepsy, the other manic-depressive insanity. It may be pertinent to remark at this point that epilepsy occurs with normality, with dementia praecox, with manic-depressive insanity; and in case No. 7, an epileptic seems to have episodes of manic-depressive insanity much like his sister, whose case is uncomplicated manic-depressive insanity. It is my belief that true epilepsy belongs fundamentally to a different group of things than either manic-depressive insanity or dementia praecox. In case No. 73, it may be seen that manic-depressive and dementia praecox occurred in different members of the same sibling group. In No. 74, as has been stated above, there is a transition from paranoid condition, which strongly resembles true paranoia, to paranoid dementia praecox and dementia praecox with feeble-mindedness.

Part II

The occurrence of both manic-depressive and dementia praecox in a group of brothers and sisters is at present a vexed question. That a parent having manic-depressive insanity may give issue to dementia praecox children seems to be established, but that a parent having dementia praecox may give issue to manic-depressive children seems to be an exceptional occurrence. For the horizontal occurrence of dementia praecox and manic-depressive, that is, in the same

generation, there are conflicting results due largely, I believe, to the varied criteria of diagnosis and to the unsettled state of psychiatric classification in general. Practically all men diagnose by rule of thumb. One or two symptoms settle the case. Hallucinations, if prominent, rule out manic-depressive for some diagnosticians. Flight of ideas rule out dementia praecox for others. Recovery or alternation of excited and depressed periods, negativism, apathy, all these symptoms which are critical for the diagnosis in the opinion of the average hospital worker are not simple matters, and mistakes occur so frequently as to invalidate far-reaching conclusions.

A very interesting side-light on this matter is found in Bancroft's (6) statistics on the relative frequency of the diagnosis of dementia praecox and manic-depressive insanity in various well-organized American hospitals. I hereby append his table, but add to it, as a last column, the percentage relationship of manic-depressive to dementia praecox as made up from his cited figures.

HOSPITAL	YEAR	AGGREGATE ADMITTED	MANIC-DEPRESSIVE	DEMENTIA PRAECOX	PER CENT MANIC-DEPRESSIVE TO DEMENTIA PRAECOX
Westborough State Hospital, Mass.	1910	491	120	121	100
Westborough State Hospital, Mass.	1912	494	115	124	92
Worcester State Hospital, Mass.	1910	568	72	194	39
Worcester State Hospital, Mass.	1912	486	77	145	53
Danvers State Hospital, Mass.	1911	573	73	150	48
Danvers State Hospital, Mass.	1912	505	92	99	93
Northampton State Hospital, Mass.	1910	330	56	66	85
Northampton State Hospital, Mass.	1912	334	57	64	88
Boston State Hospital, Mass.	1911	433	78	71	109
Boston State Hospital, Mass.	1912	651	108	97	110
Taunton State Hospital, Mass.	1911	408	37	106	35
Taunton State Hospital, Mass.	1912	520	48	172	28
Bangor State Hospital, Maine	1912	183	23	28	82
Augusta State Hospital, Maine	1912	270	55	45	122
New Hampshire State Hospital, N. H.	1911	301	56	42	133
New Hampshire State Hospital, N. H.	1912	327	65	52	125

In New York State the per cent distribution of manic-depressive insanity and dementia praecox in all hospitals was in

1911 manic-depressive, 11.2; dementia praecox, 16.0..... 70 per cent
1912 manic-depressive, 11.5; dementia praecox, 16.0..... 71 per cent

In the Boston, Augusta, and New Hampshire state hospitals there were fewer praecox cases; in all other hospitals cited, the praecox cases exceeded the manic-depressive, and in some cases the excess was over 30 per cent. The statistical variations are of such wide range as to render definite interpretation quite impossible.

With such an extraordinary variation ranging for the very low figures for manic-depressive insanity as diagnosed in the Taunton State Hospital, 35 and 28 per cent, to the very high figures of Boston State Hospital, 109 and 110 per cent, the doubt of Bancroft who wonders, "Whether the mood of the diagnostician is not reflected in the result," is too mild. It is not the mood of the diagnostician that varies so much as the diagnostic criteria of the institution. Taunton, Westboro, Worcester, Danvers, Boston, and Northampton State Hospitals are all in the State of Massachusetts, within a narrow radius of much less than 100 miles, and it is impossible that their material differs so widely as the figures would indicate.

As stated previously, one of the essential differences lies in the belief that catatonic episodes belong to dementia praecox, and the difficulty of differentiation between a catatonic episode and manic-depressive insanity. A very interesting paper by Stöcker is entitled (translation), Are there fundamental differences between a catatonic stupor and an excitement on the one hand and a depressive stupor and manic excitement on the other and in what does this consist?

To answer this self-imposed question he carries on a long comparison in parallel columns of the symptoms of catatonia and manic-depressive insanity as taken from Kraepelin's text-book. The summing up of his comparison is that the symptoms and signs in the one disease are outwardly exceedingly like those of the other, and, in fact, that an identical process is taking place, and that the difference lies in the essential original character of the catatonic and the manic. For him, as for Kraepelin, the essential defect in catatonia is the intrapsychic ataxia of Stranksy, and this manifests itself in the symptoms, especially in the conduct and speech which are without goal or plan and are dissociated from the motions, the intellect, and

the will. Whereas a relationship can be detected in manic states between conduct, speech, emotions, intellect and will. *But it becomes obvious as one reads that the differentiation is very difficult,* at times impossible, and rests on *a refinement of observation and interpretation possible only for a few men.* Indeed, one wonders whether there is such a difference fundamentally, whether every psychosis is not a dissociation, whether the acute depression or excitement of a manic is not the result of a mood or emotion almost entirely divorced from intellect, from the perception of relation; in short, whether an intrapsychic ataxia does not exist in a manic-depressive as well as in a catatonic.

(Here one may pause to state that there is no very essential difference between the intrapsychic ataxia of Stransky, the schizophrenia of Bleuler, and the psychic fragmentation or inner splitting of Urstein. Nor is there logic in grouping together in a disease entity, as each of these authors had done, those cases where such change may be found. Inflammation as a general process is found in tuberculosis, syphilis, lobar pneumonia, wound infection, etc., but we know that these are separate diseases. Similarly, the existence of schizophrenia, inner splitting, intrapsychic ataxia in very different appearing diseases does not link these cases together as belonging to the same clinical entity. The relationship may be as general as inflammation itself.)

Returning now to the subject at hand, namely, whether or not manic-depressive and dementia praecox occur in the same sibling group, different opinions are expressed in the literature. Because of the difficulties which I have outlined above, I feel that it is not incumbent on me to review the entire literature. I have selected five groups of material for consideration as bearing upon the problem.

1. The material of the Massachusetts State Hospitals. In reply to my inquiries as to whether or not manic-depressive and dementia praecox occur in the same sibling group, in their cases Danvers, Boston, Westboro, Medfield and Gardner State Hospitals reply in the negative. Worcester State Hospital reported one case occurring in two sisters. Of these sisters, one seems to be a clean-cut dementia praecox with gradual onset, depressive in character, then the later development of hallucinations, delusions of persecution, final apathy and dementia. The second sister, whose condition is diagnosed as manic-depressive, seems to have symptoms warranting that diag-

nosis from the abstract submitted to me. Northampton State Hospital reported 10 such mixed groups. It is noteworthy that this hospital makes a diagnosis of manic-depressive nearly as frequently (90 per cent) as it makes the diagnosis of dementia praecox. Moreover, most of the cases so diagnosed occur in the earlier days of the Kraepelinian scheme when recoverability and periodic insanity classified a case as manic-depressive insanity. I have not seen the abstracts of the cases from this hospital and so am unable to state further concerning them.

2. Krueger's (116) cases. Among siblings he reports only one family, No. 37, showing the union of dementia praceox and manic-depressive in two sisters. He states that the natural disposition of the sister, diagnosed as manic-depressive insanity, was obstinate and irritable. Moreover, this patient has been continuously in the institution. Certainly, it can be stated that even from the abstract submitted in this paper, a doubt as to the typical nature of the psychosis is legitimate.

3. Luther's (125) cases. Luther reports a large number of such cases; namely 11, from family group No. 82 to family group No. 92 inclusive. Of these groups, in the case diagnosed as manic-depressive in family group No. 85, alcoholic neuritis was present. Similarly, in No. 89 the original diagnosis in the manic case was hallucinatory paranoia. In No. 91, lues cerebri was the original diagnosis. In No. 92, alcoholic psychosis was entertained as a diagnosis and seems certainly to have been warranted. In 7 other cases it seems to me that atypical conditions difficult to diagnose were considered to be manic-depressive for lack of a better term.

4. Schlub's (173) material. He describes 65 families which, however, seem to have been collected from the literature rather than from his own experience or hospital material. Of these, there were 31 dementia praecox families, 17 manic-depressive families, and 12 families in which manic-depressive and dementia praecox occurred. Without going into detail concerning these 12 families, it may be stated that the diagnosis seems distinctly forced in many cases.

5. Riebeth's (158) material. Riebeth found dementia praecox throughout a family group in 40 cases, manic-depressive throughout a group in 8 cases, dementia praecox and idiocy 4 times, dementia praecox and epilepsy 4 times, dementia praecox and senile psychosis twice, scattering diagnoses in 12 families, and 14 groups in which the question of manic-depressive and dementia praecox occurring to-

gether had been considered. After studying these cases thoroughly Riebeth finds only three groups in which the possibility can be considered, and these are not clear cases. In other words, Riebeth is of the opinion that the combination occurs but seldom if at all, whereas dementia praecox is apparently related to feeble-mindedness, idiocy and senile dementia.

I feel that it would be assuming too much to state that the two psychoses do not occur together in the same sibling family group, On the whole, there seems to be evidence that they do occur. This occurrence, however, is exceptional and I am of the opinion that a typical praecox and typical manic do not often occur together. One incurs the reproach that typical cases are exceptional anyway. This I deny in the case of dementia praecox. Typical cases of dementia praecox are very common; typical cases of manic are less common, but occur. It is in the consideration of atypical manic cases around which the debate centers.

CONCLUSIONS CONCERNING THE HORIZONTAL TRANSMISSION OF MENTAL DISEASES (BROTHER AND SISTER GROUPS)

1. The psychoses of brothers and sisters tend on the whole to be alike, at least in the main characters. It is possible that the few unlike cases represent other factors than the familial traits.

2. An apparent exception is the frequency of the statement that one or other of the brothers and sisters with dementia praecox is also mentally defective. This I believe to be due to the very early occurrence of the disease, so that before it became fully manifested as dementia praecox it appeared as a more simple mental defect. This observation tends to corroborate the belief of Kraepelin that some cases of feeble-mindedness are really very early dementia praecox.

3. My own experience throws doubt on the occurrence of typical manic-depressive and typical dementia praecox in brothers and sisters. Atypical cases do occur. It is likely that in some cases the circular type of dementia praecox, with its manic coloring is involved.[3]

[3] As a result of his review of the literature Hoffmann (80) says in the case of siblings there is generally the same type of psychosis. . . . . The observation of many authors that certain psychoses, for example manic depressive and dementia praecox, do not occur in one sibling family group is not corroborated by other authors.

It will thus be seen that brothers and sisters are more alike in types of mental disease than are parents and descendants. This of course is what one would expect for there is a greater similarity biologically (and socially) between members of the same generation than between members of two generations.

Part III. Theories on the Heredity of the Mental Diseases

It is assumed that instability is a primary quality and therefore one to be dealt with by breeding it out. With indifference to the mental side of life which is characteristic of the mentally resistant class the question as to the real meaning of instability has been begged by the invention of the disastrous word "degenerate." The simplicity of the idea has charmed modern speculation and the only difficulty in the whole problem has come to be the decision as to the most expeditious way of getting rid of this troublesome flaw in an otherwise satisfactory world.

The conception that the natural environment of man must be modified if the body is to survive has long been recognized, but the fact that the mind is incomparably more delicate than the body has scarcely been noticed at all. We assume that the disorderly environment with which we surround the mind has no effect, and are ingenuously surprised when mental instability arises apparently from nowhere. But though we know nothing of its origin our temerity in applying the cure is in no sense daunted.—*W. Trotter.*

A complete consideration of the theories which underly most of the literature on the heredity of the psychoses would be a task which would lead nowhere. The major part of the literature is a repetition of the same type of work which I have criticized throughout the book (and which my own work perhaps resembles, though I have avoided the conclusions drawn by some of the writers). *Because* one believes, *in advance*, in hereditary causes of mental diseases, *therefore* any striking disease or condition in the ancestors or collaterals of a patient with mental disease is the hereditary cause of that disease—this type of thought predominates in the literature.

I. POLYMORPHISM OF MENTAL DISEASES

I have referred throughout to the writings of Esquirol and Morel (136), etc., as founders of the polymorphic theory of the heredity of mental diseases. Properly to evalute the work of these writers, which still dominates our thinking, it is necessary to remember that the foundation of it was written when the germ of tuberculosis was unknown, and tuberculosis was considered a "hereditary" disease,

when cancer and the like diseases were entire mysteries and no transplantations of carcinomatous tissue from animal to animal had been made, when the syphilitic origin of general paresis or of tabes dorsalis was unsuspected. Modern psychology was almost unborn, and the endocrines were unsuspected except by Addison and Brown-Sequard. Even neurasthenia had not been studied when the French psychiatrists formulated their theory. Stains for studying post-mortem tissue were crude and of little importance, and no one studied scientifically the blood constituents or its chemistry, and even the knee jerks had not been discovered. Great as were Esquirol and Morel it is folly to expect that their far-reaching generalizations are really valid today. Yet in one form or another they are back of most of what is said concerning the heredity of mental diseases.

There are the following cardinal points in the theory as elaborated by many writers and tacitly accepted by the majority of psychiatrists, biologists and eugenists even today.

There is a unitary something in the neuropathic or psychopathic inheritance which makes itself manifest under many forms (thus the *polymorphism*). All manner of mental diseases, including the organic diseases, are thus labelled, and all psychoneuroses, including as well epilepsy, feeble-mindedness, and crime. Up to this day this doctrine prevails in France, in England, and to a limited extent in Germany where attention to the details of mental diseases has side-tracked polymorphism and in America where under a new guise, as Mendelism, it has been given the sanction of the foremost writers until very lately. In the excessive development given this theory headache in an ancestor was given a hereditary value in relation to the mental disease of his descendant. Even headache and fainting spells in a cousin had a dread significance, while the earlier writers laid great emphasis on tuberculosis, cancer, hemiplegia, gout, "rheumatism" and every chronic or semi-chronic human ailment. At the present day practically every mental hospital history in America wastes good paper by including data on these diseases in relation to the ancestors and relatives of patients.

Lombroso (124), and his pupil Nordau (146) gave to the term degeneracy the widest significance, so that on the one side the lowest human beings—cretins, idiots, etc., were linked together with the men and women of genius. His "reasoning" is typical of that of the whole polymorphic school and I cite a few examples of it, though

I could multiply them indefinitely in the writings of Morel, Fere, Dejerine, Clouston, MacPherson and others who have subscribed to the theory.

Lombroso based much of his theory on his ideas of the stigmata of degeneracy which are both physical and mental. Thus he gives as signs of degeneracy common to both genius and insanity shortness *and* tallness, rickets (which includes, God save the mark, rachitic, *lame, hunch-back,* or clubfooted, thus linking together at least a hundred diverse things under one heading), pallor and emaciation (which as every one knows are secondary to other conditions), stammering, left handedness, delayed development, precocity, etc., up to a "fondness for certain words!" And this passed for science, and was part of the work of an extraordinary personage. It never occurred, apparently to Lombroso that all these stigmatas are very common and found almost as frequently among the "normal" as among the insane or the geniuses. He finds it rather sinister that a list of geniuses were weak and sickly in childhood, such personages as Demosthenes, Bacon, Descartes, Newton, etc. It would surely be a blessing if more people were then weak and sickly in childhood, if, alas, mediocrity did not also follow childhood diseases.

In another place he notes the greater capacity of the skull in men of genius, and likens it to the "case reported of an insane man with a large skull." As a matter of fact, the weight of the brain in insane hospital autopsies is certainly not greater and is generally less than that observed elsewhere. "Men of genius frequently stammer"—I know many stammering mediocrities. It is hard to believe that Lombroso's work found so great a vogue, so patent are its absurdities. Thus he lays stress on the *resemblance* of monomania to the distinguished pertinacity by which men of science devote themselves to one problem!

As a matter of fact amongst the 700 families of the insane at Taunton I found no cases of "genius" and few of high-grade talent. Yet Lombroso concludes "we may confidently affirm that genius is a true degenerative psychosis belonging to the group of moral insanity and may temporarily spring out of other psychoses." All the evidence he produces is to adduce *resemblances* of the flimsiest kind, without any "control" of "normal" families. In fact his definition of degeneracy as anything deviated from the commonplace must inevitably force genius into degeneracy, since the very essence of

genius is that it is peculiarly exuberant in certain qualities. Thus the mistake in logic which nullifies all the work of the polymorphists is seen at its best in Lombroso, i.e., to make the premiss inevitably include the conclusion, to "prove" what one assumes.

What we do know of the mental diseases, with the exception of the organic diseases, is little enough, and it is only on the basis of that ignorance that we can lump them together. What we do know of "epilepsy" is that its causes are many; what we do know of neurasthenia and the like psychoneuroses is that often enough their origin can be traced to infections, injury, emotional tension and the circumstances of life, while the uncertain factor we call heredity is the point to be proved. Indeed, it is true that the "psychoneuroses" are often merely minor mental diseases, but this need not obscure the entire situation for us. What we really know of criminality is that it is no entity, that the same crime may be committed by entirely different personages.[4]

[4] I have recently examined a half-dozen of murderers. One was an Italian, brought up in Sicily where it is commonplace to kill in a quarrel. In a drunken fight his knife reached into the vitals of his opponent. Another was a Negro, a lodging-house keeper, who shot and killed a policeman in a gun battle following an aggression by the officer. The third was an Irish boy, who killed with an axe his successful rival for the affections of a girl. This lad was undoubtedly psychopathic, heard voices, and attempted suicide. The fourth was the black sheep of a good family, a delinquent from childhood—thief, swindler, gun-man—cruel and without a grain of human feeling, a monstrosity in his lack of social feeling. The fifth was a chronic alcoholic who killed in a frenzy of alcoholic jealousy. The sixth was a woman, deserted by her lover, thrown out from her apartment by her successor, who thereupon shot and killed the man. Certainly the first case was an accident of racial culture, the second pathological only in its social setting, the third was psychopathic, the fourth presents the problem of crime and the real criminal, the fifth is alcohol and its ramifications in mental pathology, and the sixth is the sex question and its social pathology. It would blur all the essential difference to label all these murderers as psychopathic, and it would defeat the pursuit of real knowledge and real treatment.

A classical situation has been depicted by my friend, Dr. A. Warren Stearns (184). A group of four young men acting as a gang rob a store. The gang is composed of a college graduate of superior intelligence, a high school graduate, a young mechanic of good training and ability, and a feeble-minded man of 25. The records of these men would not in the least indicate which was feeble-minded and which was not. To lump criminality as psychopathic is to forget the teachings of history, which show that crime is in largest part a social matter, and to throw overboard everyday commonsense.

Féré's, La Famille Neuropathique is as typical an account of polymorphism as is found in the literature. What I call clinical blurring is found at its best in his pages, *though it is to be remembered that for his times his conceptions were advanced.* But when a modern clinician puts together convulsions, hysteria, idiocy, epilepsy, strabismus, paralysis, neuralgia, cerebral inflammations, deaf mutism, exophthalmic goitre, gout, tuberculosis, paralysis agitans, etc., as neuropathic; when he totally neglects the splendid researches which show that convulsions has a dozen sources, that exophthalmic goitre is not neuropathic but endocrinopathic, that apoplexy may be caused by syphilis, tumor, or cerebral arterio-sclerosis; that paralysis agitans is a disease of the lenticular and other nuclei possibly of substantia Nigra and is in the case of epidemic encephalitis definitely associated with a known infection; that tuberculosis can be produced in any guinea-pig (whether neuropathic or not) by injection of infectious material, then we have a right to discount entirely his conclusions as to the heredity of mental diseases. Féré, his contemporaries and predecessors, were brilliant men—unfortunately they translated resemblance into identity[5] and boldly generalized generations beyond the status of their times in knowledge. Today even we are far away from any "laws" in these matters.

II

The second point in the polymorphism theory follows quite naturally from the polymorphic idea. There is a transformation from generation to generation of the mental or nervous disease, a transformation proceeding from bad to worse and finally in four generations leading to race extinction. In the classical scheme there was a sort of inverted hierarchy of degeneration which started in the first generation with character peculiarity of one sort or another—

[5] This translation of resemblances into identities is illustrated in Féré, as in many other writers. Thus he considers migraine as a sensorial form of epilepsy, and also includes asthma, and certain forms of tic as epileptic. A colleague of mine goes a bit further and includes, as epileptiform, *outbursts of temper.* It is curious that the *periodicity* of the menstrual period has not lead to its inclusion in the epilepsies.

In fact the essential feature of epilepsy is loss of consciousness in an explosive or sudden manner. Migraine is usually of deliberate evolution and never associated with loss of consciousness.

miserliness, spendthriftiness, genius, bad temper, pomposity, and a curious mixture of all kinds of qualities, some the very opposites of others; which manifested itself in the second generation with the recurrent depressions and excitements, involution psychoses, toxic-exhaustive states, alcoholic mental diseases, drug intoxications, appeared in a third generation as the dementing psychoses, epilepsy, etc., and in the dismal fourth generation as cretinism, Mongolian idiocy, idiocy in general, monstrosities of one type or another, reaching race or stock extinction.

It is curious that none of the writers has any especial evidence for this schematic transformation. Of course they cite plenty of examples where these various conditions succeed each other for a generation or two, and the literature is full of the eccentric father who begot the insane son, or the relation of epilepsy in one generation to the insanity in the previous generation. The only four generation family that I find definitely known is that which I have cited where mental disease of approximately the same type appeared, and where but little essential change was noted. In the other families cited one observed a moderate swing toward dementia praecox from what seem milder diseases, and also the appearance of feeble-mindedness or at least mental defect is noteworthy. But it is also evident that the worst types of mental disease arise without any preliminaries in the previous generations; it is also evident, e.g., that Mongolian idiocy needs no psychopathic background for its origin, epilepsy arises de novo, as does much of feeble-mindedness.[6] Also one finds "normal" descendants of the insane, people who succeed, obey laws, act and look normal. By including *all* the collateral relatives of any case of mental disease one could make something of a case for the transformation theory but that would beg the main question which is, whether headache or miserliness in a great aunt has anything to do with dementia praecox in a patient. If it has then it condemns all of us to a psychopathic career.

[6] Indeed it stands out that the worst cases of feeble-mindedness—the cretins, the mongolians, the deplorable blasted idiot of lowest grade, are not the end product of four generations of psychopathia, but on the other hand, arise in the midst of an otherwise normal generation from normal ancestors. Contrariwise, the moron, the milder feeble-minded case, is, so far as my own research at the present time shows, likely to be the member of a defective generation and come from a defective stock, in which, however, no transition of psychopathic type occurs.

Parenthetically, it may be stated that "the fallacy of the positive instance" appears in vicious form throughout the psychiatric literature, and especially in relationship to the inheritance of mental diseases. The early authors cite only the cases in which a mental disease in a descendant follows some abnormal situation in an ancestor. The negative instances where an abnormal situation in an ancestor brought no mental disease in a descendant, was not considered by them at all, but this surely is necessary to exclude coincidences. In a sense, it brings all such work on inheritance of mental diseases in the category of pseudo-science, according to the dictum of Oliver Wendell Holmes, who stated that you may know a pseudo-science by the fact that it cites only the positive instances and never by any chance shows any scepticism towards its own results or conclusions. Furthermore, there is a fallacy in classing as the same kind of thing, and necessarily as heredity, the cases where mental disease occurs from generation to generation and those cases where it occurs only in members of one generation in what have hitherto been sound stocks. It is conceivable that there is necessarily a germplasm alteration to account for the mental disease that is present in parents and descendants, but it may well be that where a mental disease is present in several brothers and sisters that we are dealing with some change in the uterine environment which affects them all, or some direct blastophoric influence. In other words, the burden of proof of a germinal alteration in the case where only the individuals of one generation are involved, rests upon those who affirm it, and not upon those who would assume an environmental situation of some type or other.

On the whole the most recent German work rejects at least in its totality and in its scheme the theory of polymorphism. Strohmayer (191, 192, 193) throws out of court entirely the work done by the French investigators and that of their followers, stating that the statistics which have been gathered merely declare how frequently the various diseases mentioned can be found in the ascendants and collaterals of the patients, but give no idea whatever as to a causal connection. He points out also that practically no one of his predecessors (and in fact practically none of his successors) have paid any attention to the sound members of a tainted family. On the ground of their investigation Diem (46) and Jolly (100) opposed polymorphism. Wagner Von Jauregg (94) is especially trenchant

in his denunciation of the type of work done to substantiate polymorphism. In the earlier days of his writing Kraepelin favored polymorphism. In the later editions of his text books he takes the stand that where it appears we are not dealing with a true heredity but with germplasm injury (blastophoria). Bleuler (17) in his text book pays little attention to heredity as a cause, and states of the theory of polymorphism that it can be substantiated only if one spreads the relationship far and wide, and embraces many conditions as being neuropathic. Crouse (83), Bischof (15), Kreichgauer (114), Frankhauser (61), Hoffman (83, 84), Witterman (218), Riebeth (158) and Rudin (164, 165) may be mentioned as having published studies of groups of families, and who oppose polymorphism. There have been, of course, a few German workers who have believed in polymorphism, of a type, notably of the later writers, Schuppius (174) and Luther (125). On the whole the authors of the really clinical studies are opposed to polymorphism, whereas those who lump together in an indiscriminate way all mental diseases, all nervous diseases, and almost all abnormalities of any kind, favor, it, naturally. Says Hoffman (83), "The conception of a similar predisposition for all psychoses is on logical grounds to be rejected. So long as we recognize clinically different disease complexes as different diseases we must lay down as a basis different predispositions. . . . . For the degeneration scheme which has been drawn up by various authors there are absolutely no trustworthy empirical facts." Reviewing the entire literature, as does Hoffmann with a clear eye, he comes to a definite conclusion that polymorphism is to be rejected both as hindering the growth of knowledge concerning the inheritance of mental diseases, and because there is no evidence of trustworthy kind for its existence. I recommend without reservation to every reader interested in the inheritance of mental diseases the remarkably thorough work of this writer.

Though polymorphism as a scheme and in its broadest form is to be rejected, it nevertheless remains true that in a very limited form there is some basis for it. There is a basis for the belief that in many families a disease in an ancestor is followed at an earlier time and in a more severe form in a descendant, and that further, as I have repeatedly stated, a mild psychoneurotic type of a mental disease in a descendant. There *seems* to be a tendency towards dementia praecox in the descendants of ancestors suffering from

such mental diseases as manic depressive insanity, the involution psychoses, and the senile dementias. Further, it is not uncommon to find a certain type of mental defectiveness in the descendants of dementia praecox patients. In this limited way a certain type of polymorphism may be observed. On the other hand, there is not the slightest doubt that the descendants of a person with mental disease may be much better than he, mentally may progress towards normality, and that all kinds of transitions and haphazard half-way stops may be noted.

MENDELISM AND MENTAL DISEASES

What is here stated as to the value of the Mendelian laws in regard to the inheritance of mental diseases in no way refers to the work of the illustrious Gregor Mendel or to the brilliant experimental work in botany and zoology of his followers. That there are unit characters, that these appear and disappear in mathematically analyzable fashion seems undoubted, at least for the simple characters. It is to the entrance of enthusiastic Mendelians into the field of psychiatry and their conclusions that I direct the adverse criticism which has been apparent in other parts of this book and which again I formulate.

a. The mental diseases as due to the lack of a unit determiner which is present in the normal (Davenport (39) Goddard (66), and the American Mendelians).

In reality this is merely a polymorphism with a Mendelian explanation, based on a statistical analysis. If one denies that it is at all proven that there is a unity equivalence in the diseases of a list which starts from A, proceeds alphabetically to include apoplexy, alcohol, blindness, etc., etc., down to tumor, then it is obvious that one must deny the validity of the conclusion. It is good to be bolstered in this attack upon the work of so eminent a man as Davenport by the words of a Conklin.

Recent studies have shown that the development of such simple characters as coat color (Wright), eye color (Morgan) and sex (Wilson, Lillie, Goldsmidt) are exceedingly complex and that very many hereditary factors may be involved in the process. When we come to the development of more complex things such as temperament, feeble-mindedness, insanity, personality, we are dealing with the most complex phenomena in all the world—inconceivably more complex than any of the problems of astronomy, physics or

chemistry. If eye color in the fruit fly is dependent upon a large number of inheritance factors, as Morgan and his pupils have shown to be the case, how much more probable is it that epilepsy, feeble-mindedness, genius and insanity are dependent upon a still larger number of inheritance factors, as well as upon an innumerable number of environmental causes? We may be sure that when the whole "alphabet of degeneracy from alcoholism to wanderlust" is attributed to the lack of a single hereditary factor, there has been a pitiful failure to recognize the complexity of the phenomena in question.[7]

Even if it were true that all of these complex mental and physical states were evidences of a unitary defect the method by which the data were gathered would in itself invalidate the conclusion. I have the highest respect for social work, and I have employed field workers whose labors I respected, but I repeat, it is impossible for any one non-medically trained to make conclusions concerning the mental and physical states of the dead on the basis of an interview or a court record, or to reach a diagnosis in the case of the living by a face to face interview. I would not trust as adequate the field work of a trained psychiatrist, working in the usual manner, viz., asking questions from relatives, friends and enemies. Such work is not without value, and we must use it at present, but it is too slender to bear the weight of a mighty Mendelian conclusion.

It may seem that I am unduly severe in my criticism. Therefore, I quote from David Heron (77), whose criticisms are those of Pearson and the biometric school. He states as follows:

> Probably nothing during the course of the last twelve months has been more detrimental to the true progress of Eugenics as a science than the papers read and the statements made at the recent International Eugenics Congress. In some branches of knowledge little harm would be done by the statement of erroneous conclusions; no public opinion would be based upon them, and there would be no likelihood of social action resulting from them. But with Eugenics the matter is wholly otherwise; racial improvement and racial deterioration interest, and rightly interest, large masses of our citizens, who have no means of ascertaining whether a given statement is or is not a great scientific verity. They see it stated in the daily newspapers that Professor So-and-so, at what they take to be a great congress of specialists, has declared that degenerate stocks mend themselves by dying out; or they hear that Dr. Blank has discovered that insanity or mental deficiency obeys the laws of Mendel, and that the children of an insane parent, if normal themselves, may marry other normals without any detriment to their future offspring or the race.

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[7] Development of Personality, Edwin Grant Conklin, p. 126–127.

I quote the next few paragraphs as indicating the method used by Davenport and his associates in collecting data, to show in how thoroughly prejudiced a manner the data quoted, and how the results found are almost surely forecasted in the technique.

Clear evidence as to the bias with which the data have been collected is afforded by a study of Bulletins Nos. 2 and 6 of the Eugenics Record Office. In Bulletin No. 2, "The study of human heredity; methods of collecting, charting, and analysing data," by C. B. Davenport, H. H. Laughlin, D. F. Weeks, E. R. Johnstone, and H. H. Goddard, the methods "in use at the Eugenics Record Office" are stated. On page 7 et seq. we find the following:

"Limits to pedigree. How far among collaterals is it desirable to extend the pedigree? This depends on the nature of the primary trait. . . . . So many traits are inherited in accordance with the Mendelian rules that a brief statement of them is appended. But the field-worker is warned against being so prejudiced by these rules that her, or his, judgment is warped. . . . .

"Some defects that the field-worker will study, such as albinism and feeble-mindedness, are known (!) as recessive defects, i.e., they are defects due to the absence of the determiner making for normality in respect to these traits. Other defects, such as cataract and brachydactylism, are dominant defects, which means that they are due to the presence of some germinal determiner in addition to all the determiners for normality in respect to these characters. . . . .

"For example, by hypothesis, feeble-mindedness is for the most part a recessive trait, and the hypothesis must be tested as follows: The field-worker finds a person suffering from feeble-mindedness, a descendant of two normal parents—by hypothesis both of these parents are simplex; the field-worker must understand that each parent will probably have somewhere in his or her ancestry a feeble-minded person, and it is the business of the field-worker to make a special search for such person or persons in the pedigree."

It is difficult to understand how the field-workers would fail to be "prejudiced by these (Mendelian) rules" when they are instructed "to make a special search for the person or persons" who are considered necessary for the support of the Mendelian theory.

The polemic of Heron goes on remorselessly to show that the great number of contradictions found in the writings of Davenport and his associates to show that the expected ratios are not fulfilled even when the technique of collecting has been as above. Heron is scandalized, as he should be, by the lack of knowledge of disease shown by the American eugenists, who lump together "cases of unchastity, hysteria, simple meningitis, apoplexy, paralysis, and alcoholism."

It will, we think, be clear that no legitimate conclusions whatever can be drawn from data collected under these vague categories which admit of any amount of personal equation, and which are then tabled in this slipshod manner. We may safely say that no proof of Mendelism applying or not applying to mental defect can be deduced from such work, and the manner in which these memoirs have been accepted as valid contributions to science by responsible Mendelians and Eugenist publicists in this country is not only disastrous for Eugenics, but does grave harm to Mendelian theory itself. We know no more about the heredity of feeble-mindedness and epilepsy than we did before—we know only the central fact that these defects are certainly hereditary; and considering the haste and carelessness with which these pedigrees have been collected and published, we very much doubt whether this central fact has received any solid basis of additional demonstration.

In a tone fully as rancorous as that of Heron, A. J. Rosanoff (162) has responded to the attack on the American Mendelians. He begins his defence by an attack on the "so-called investigations of heredity" carried out by the Pearson English school of biometricians. He then cites as a defence of the inclusion of all kinds of defects as unitary, the fact that eminent psychiatrists believe this to be true and cites, e.g., amongst others the statement of Peterson who says in his text book article[8] that in determining the factor of heredity we must not be content with ascertaining the existence of psychoses in the ascendants but must seek by careful interrogation of various members of the family for some of the hereditary equivalents such as epilepsy, chorea, hysteria, neurasthenia, somnambulism, migraine, organic diseases of the nervous system, criminal tendencies, eccentricities of character, drunkedness, etc., for these equivalents are interchangeable from one generation to another and are simply evidences of instability of the nervous system. It is the unstable organization that is inherited, not a particular neurosis or psychosis, and it must be our aim in the investigation of the progenitors to discover the cause of this.

Here is the frankest possible polymorphism, a pure assumption from beginning to end, a complete unification of all kinds of entities and non-entities, a conclusion vitiated by the fact that it is contained in the premiss, etc. Rosanoff has not bettered the cause of the Mendelian inheritance of the psychoses, etc., by his use of authorities.

[8] Church and Peterson, Nervous and Mental Diseases.

Further, normality is balanced against this psychopathy as a unitary something, to be discovered by learning, e.g., that a man seems intelligent and pleasant, works steadily, dresses fairly well, and has managed to keep out of jail and the lunatic asylum. But only the one wedded to a theory, or naïvely uncritical, can believe it is a real unitary something. Normality is an abstraction like the Average Man of the statistician, or like Truth, Charity and Justice. There are hot tempered "normals" and phlegmatic normals, there are brilliant folks and dull ones, there are those whose sex-desires. run riot in their hearts but who control conduct, as there are those whose desires running as vigorously just lack control. The disharmonies of the average man are almost infinite in variety but remain within the degree necessary for the average of conduct. Some one not essentially different may have just disharmony enough to be a good misfit in life. Often enough the difference between normality and abnormality is the quantitative difference existent between an unsaturated solution, which is clear, and one which having but a few grains more of a salt, is saturated and consequently turbid. The totally dissimilar appearance of clearness and turbidity depend then, not on qualitatively differing causes but on a quantitative relationship. One may be normal in conduct and appearance who merely lacks courage to be otherwise, just as one may be abnormal who lacks some instinct or inhibition or who possesses to excess, and in a disharmonious way, a quality.

The most serious criticism that is to be made against the Davenport School is that it postulates Mendelism in advance, considers all its researches only in the light of this theory, and dismisses entirely all other conceptions. It is well to have a working hypothesis, but when the hypothesis is made of primary importance then indeed it hinders rather than helps science.

MENDELISM AS APPLIED TO MANIC-DEPRESSIVE AND DEMENTIA PRAECOX

Several authors have studied manic-depressive and dementia praecox in relationship to the Mendelian laws, discarding the fallacy which underlies work that lumps all mental diseases together. The various authors have sought to discover Mendelian laws in relationship to isolated mental diseases. This is a far more logical procedure and has been carefully worked on by Rosanoff in this country and by Rudin (166), Jolly (100), Wittermann (218), Luther (125),

Krüger (116), Hoffmann (84), Weinberg (211, 212) Lenz (121), and others. I refer the reader for a review of this work to Hoffmann's article, and also for a statement of Mendelism as applied to mental diseases. It would lead me too far afield to give the possible Mendelian matings, the interrelationship of the homozygote and heterozygote, of dominant and recessive characters, in this volume. Rudin, who has worked according to approved statistical methods formulated by Weinberg, makes only one really positive statement, viz., that certain forms of dementia praecox in certain families follow the recessive type of inheritance, and he states very soberly later on that no one can, on the basis of researches up to the present time, state that Mendelian laws *cannot* apply to the inheritance of mental disease. That is, though it is apparent that he wishes to find definite Mendelian laws, this candid worker does not do so to any extent. On the other hand, Rosanoff and his associates lay down quite definite relationships making normality dominant to manic depressive, that dominant to dementia praecox, and this dominant to epilepsy. Jolly believes that manic depressive is not a simple dominant character, although he believes that schizophrenia is a simple recessive character. Krüger and Schuppius, Witterman and others have also investigated the problem, with meagre positive results and much skepticism concerning their own findings. It seems to me that we are very far from that stage in the knowledge of mental diseases where we can seek to apply laws which apply at the present time only to the simplest biological characters, and which when applied to complex characters lose their entire Mendelism.[9]

[9] "Most students of genetics realize that a factor difference usually affects more than a single character. For example a mutant stock of Diosophilia called "rudimentary wings" has as its principal characteristic very short wings. But the factor for rudimentary wings produces other effects as well. The females are almost completely sterile while the males are fertile. The viability of the stock is poor. When flies with rudimentary wings are put into competition with wild flies few of the rudimentary flies come through especially if the culture be crowded. The hind legs are also shortened. All these effects are the results of a single factor difference."

The above quoted from T. H. Morgan (cited by Holmes) contains to me an incomprehensible conclusion. Just why is it stated that the principal difference is the rudimentary wings when there is sterility, lowered viability and short hind legs as well? And how is the conclusion of single factor difference reached? In the early Mendelian work the emphasis was on outward characters easily studied and recorded, to explain which hypothetical gam-

That is to say, when the effort has been made by biologists to apply Mendelian laws to the complex characters the result is a subtlety of discussion and a hair-splitting which makes one think that one is concerned in some discussion of the schoolmen. We have established no pathology for manic-depressive or dementia praecox. We have no absolute criteria for their diagnosis. We do not know whether they are a dozen characters rolled in one or whether they are mere diseases. How then apply Mendelian laws to their occurrence in families? Further, there are certain statistical factors which would make it impossible to discover Mendelism in these diseases. To discover Mendelian laws in relationship to any mental disease or other human character, it would be necessary to discover Mendelian statistical ratios. Obviously these cannot be found in mental diseases. In the first place the infant death rate militates against it. We do not know of any child who has died early in life whether or not he would have had mental disease when he grew up, since mental diseases of the manic depressive and dementia praecox type are diseases of adolescence and adult life. Further, miscarriages which are so common would have the same damaging effect upon the statistics. Again the prevention of childbirth, which is so prevalent in the last generation, knocks out of the possible any Mendelian ratios, because we do not know whether or not the possible children would have been well mentally or sick. It may be true, as Rudin says, that no one has a right to say that Mendelian laws may not apply to the mental diseases. It is also true that most of the Mendelian work done in mental diseases has either corroborated an already formed conclusion or else seems like a mathematical exercise in ingenuity illustrated by interesting formulae. The future may show that Mendelian laws govern the inheritance of mental diseases—at the present time there is nothing to prove this.[10]

etes were evoked. Now the shift is to speak of the invisible factors as if they were in the same demonstrable plane as the color of a pea-pod or its contour. Back of the rudimentary wings, etc., may be a lowered viability of germplasm rather than any factor difference.

[10] For a criticism of the Davenport work and that of the earlier work of Rosanoff and Orr, see Weinberg and Hoffman. In their earlier work Rosanoff and Orr followed Davenport's scheme of postulating the neuropathic character as minus a unit determiner, unifying under this psychopathic disposition all the psychoses, also convulsions, alcoholism, irritability,

THE BIOMETRIC SCHOOL AND THE HEREDITY OF MENTAL DISEASES

I have not discussed the work of the biometricians—Pearson (154), and his followers. The eminent English scientist and statistician has followed a very simple scheme for studying the inheritance of "insanity." His method is as follows (I quote from his article, On the inheritance of insanity). In any disease:

1. Ascertain the prevalence of the disease or character in the population at large.

2. Take a sample, say of a thousand persons with the disease and record the incidence of the disease among their ancestry and collaterals.

3. Take a sample, say of a thousand persons without the special disease, and investigate the distribution of the disease among their ancestry and collaterals.

He then takes Diem's figures (an insufficient number) and putting together "as want of mental balance" the alcoholism, insanity, senile dementia, eccentricity and suicide of the parents and relating it to the insanity of the descendants Professor Pearson finds a coefficient range of 0.25 to 0.30. He then compares this to the coefficient range of those human characters found by his statistical method and the Galtonian inheritance scheme to be inherited, viz., stature, span, cubit, eye color.

	Parental inheritance
Stature	0.51
Span	0.46
Cubit	0.42
Eye color	0.49

etc. Both Hoffman and Weinberg agree that no such unification of psychopathic situations can be made, and severely criticize from the statistical standpoint both the figures and the method by which they have been collected. I quote Hoffman, (page 281) "The main defect of this work (he refers to the work of Davenport and Weeks) lies in other directions. In the first place there is no good ground which brings together epilepsy and feeble-mindedness, and which allows us to make a unity of different things or to make common laws for them." Further, in relation to the appearance of epilepsy in the children of psychically healthy parents it is to be remembered that all kinds of exogenous factors come to hand without the necessity to invoke a common constitution for epilepsy. The same is true for the different forms of feeble-mindedness. Under these circumstances it would seem that these two anomalies at least are hardly suited for research according to Mendelian laws.

He concludes that there is a great degree of inheritance of insanity.

When a statistician seriously compares the simple qualities of stature, span, cubit, which may be measured with a ruler, and eye color which any one not color blind may decide upon at a glance with the conglomeration and protean mass of alcoholism, insanity, senile dementia, eccentricity and suicide, and seriously asks for respectful consideration of his conclusions, then there is nothing respectful to say about statistics. When it is remembered that the statistician is one of the world's great writers and a power in the world of thought then it becomes necessary to say that psychiatry is not yet ready for the aid of statistics in the solution of such problems as the heredity of mental diseases. Not that the statistician is really to blame if he drops critical frame of mind of science when he enters the field under discussion. The maleficient words insanity and heredity apparently exercise a baleful influence on the mind.[11]

The fact is that the inheritance of mental diseases is a clinical problem to be solved as any other clinical problem is solved by pathology and individual study and experiment. Until we have facts to offer for statistical analysis the statisticians and with them the professors of biology had better keep out of this field. Both statisticians and biologists have helped keep up interest in the heredity of mental diseases and they need not attack one another for both groups have been honest and well intentioned. But there is a classical proverb concerning good intentions.

Part IV. Weismannism and Blastophoria

That which is an advance may, if it becomes a dogma, retard the growth of science. This seems to me to have been the case with certain phases of that form of Neo-Darwinism called Weismannism.

Weismann (213) seemed at first, and until lately, to have answered the question once and for all, whether or not acquired characters are inherited, by a definite "no." His doctrine of the continuity of

[11] S. E. Jelliffe (96), "Predementia Praecox—Hereditary and Constitutional Features of the Dementia Praecox make-up." "We include the work of the biometricians themselves within this criticism. Those working in a strange field have been unduly influenced by a term—insanity—and have approached the nonsensical in their attempt at analyzing so many diverse conditions grouped under a legal rather than a medical concept."

See this writer's work for a caustic criticism of the "heredity" work which fills the literature.

the germplasm and its independence of the somaplasm, in fact, the dependence of the latter upon it, made a profound impression upon the thought of our times, and crowded into disrespectability Lamarck's theory that new characters arose through use and disuse, through direct response of living creatures to environment and the inheritance of these changes. Though Weismann did not deny and in fact conceded that the environment might act to alter in an injurious way the germplasm he made the germplasm so superior to somaplasm that the latter became very unimportant in the eyes of biologists. But while germplasm may be theoretically and practically the most important thing under the sun, somaplasm is the individual, it is the sick man we physicians must consider, it is the inmate of hospital and jail, it is the face at least of the problem. Nay more, it is the heart and soul of the problem, for while germplasm is important it is so only in the light of the somaplasm it may produce. The individual may be, as is so often asserted, a mere accident in the history of the germplasm, but if so he is the accident with which mankind deals and whose welfare constitutes the value given to things and to germplasm.

"The body develops and dies in each generation; the germplasm is the continuous stream of living substance which connects all generations. The body nourishes and protects the germ; it is the carrier of the germplasm, the mortal trustee of an immortal substance." So says Conklin, and in some respects what he (and other biologists in less eloquent terms) say, is true, but in other respects it is rather more poetical than scientific. For if germplasm is to remain eternal the merely mortal individual must unite himself with another merely mortal individual, and their parts must be so adapted that sperm (carrier of male heredity line) may meet, in a proper environment the ovum (carrier of female heredity line). If there be an acute anteflexion of the uterus or a gonorrheal stricture of the urethra the immortal germplasm will die with the individual who "clothes it," "genotype" to use Johannsen's terms will die with "phenotype." A thousand and one things may kill the immortal germplasm, from syphilis and a carbolic acid vaginal douche to the domestication which destroys the fertility of wild animals. Further supposing male and female unite and *are* fertile, i.e., germplasm carried in sperm unites with germplasm carried in egg, the resulting germplasm of the descendants is neither one nor or the other, it is a new product though

it may have the characters of both. Unless we are talking of the germplasm of the entire race there is no immortality worth talking about.

Conklin (30) himself points in trenchant fashion an important mistake in Weismannism at least as it is usually interpreted.

> But important as this distinction is between germ and soma it has sometimes been overemphasized. This is one of the chief faults of Weismann's theory. The germ and the soma are generically alike, but specifically different. Both germ cells and somatic cells have come from the same oosperm, but have differentiated in different ways; the tissue cells have lost certain things which the germ cells retain and have developed other things which remain undeveloped in the germ cells. But the germ cells do not remain undifferentiated; both egg and sperm are differentiated, the former for receiving the sperm and for the nourishment of the embryo, the latter for locomotion and for penetration into the egg. But while the differentiations of tissue cells are usually irreversible, so that they do not again become germinal cells, the differentiations of the sex cells are reversible, so that these cells, after their union, again become germinal cells.

He even goes on further to state that germplasm is not limited in distribution to the cells of generation, it penetrates into every cell of the body as the chromatin of its nucleus.

> Chromatin is germplasm, cytoplasm is somatoplasm. In many theories of heredity it is assumed that there is a specific "inheritance material," distinct from the general protoplasm, the function of which is the "transmission" of hereditary properties from generation to generation, and the chief characteristics of which are independence of the general protoplasm, continuity from generation to generation and extreme stability in organization. This is the idioplasm of Naegeli, the germplasm of Weismann. Such a substance is no mere fiction or logical abstraction, as many writers have affirmed, for there is in the nucleus of every cell a substance which fulfills all of these conditions, namely, the chromatin. It is relatively independent of the surrounding cytoplasm, it is self-propagating and consequently continuous from cell to cell, and from generation to generation and it is relatively stable in organization so that it is but little influenced by environmental conditions. There are many important reasons for believing that the chromatin is the germplasm, or at least that it contains the inheritance units, as we shall see later. It is present not only in germ cells but in every cell of the organism, though in highly differentiated tissue cells it may undergo certain secondary modifications. On the other hand the cytoplasm surrounding the nucleus, undergoes many marked differentiations in the course of development and it constitutes in the main the body plasm or somatoplasm. Germplasm and somatoplasm are not, therefore, vague generalizations, but they are definite cell substances which may be seen under the microscope.

Yves DeLage (44) indeed goes so far as to state quite definitely that the difference between germplasm and somatoplasm is the difference between undifferentiated cell and differentiated cell. In this theory, which will be given in more detail further, it is the ability of undifferentiated cells to multiply indefinitely which is characteristic of germplasm. Further, he shows that somatoplasm can also create germplasm. Thus in the potato, the non-germinal parts if planted can reproduce the entire potato plant, and other bulbs have the same property, not to speak of certain of the lower forms of life which may be cut in parts and each part reproduce a perfect individual. The reason that germplasm is relatively immortal and somatoplasm is not, says De Lage, is because the latter is so differentiated and in such intimate relationship with the rest of the body that it has lost property of growth, which is the first condition for reproduction. Says DeLage, "The developed individual is the product of many factors, all equally indispensable and important. The germplasm is only one of these factors. The others are tropisms and tactisms, functional stimulation, the action of nutrition in relationship to the ingested materials and to the excreta, and varying conditions of all kinds."

The celebrated pathologist, Carl Weigert (210), although he adversely criticizes Naegeli's idea that there is in every body cell a reticular substance which is idioplasm (or Weismann's germplasm), also criticizes Weismann for so sharply differentiating body and germ. Weigert believes that there are intermediate tissues between the undifferentiated germplasm and some of the highly differentiated tissues, that as a tissue grows more differentiated it is more unlike germplasm and as it is less differentiated it is more like germplasm. In other words one of the cardinal tenets of Weismannism, the complete separation in qualities of germplasm from body plasm, is not agreed to by Naegeli, De Lage, Weigert, and Conkln, to say nothing of many others.

The fundamental question for most mortals is not whether acquired characters are inherited in the strictest sense of the term, it is as Schallmayer asks "Can the experiences of the phenotype alter the genotype?" Can what happens to the individual alter the germplasm and thus alter the next generation? It is not necessary for those of us who are medical men that this alteration persist as a new character, forever—that may be the only important question

for biologists who deal with, to them, unimportant animals or plants, but for us who deal with humans *any* group of human beings thus altered is important.[12] Each generation is the human race of that time, its peculiarities and failings become impressed on future generations, not only through germplasm but through laws, customs, economic conditions and so forth, which are at least nearly as important as germplasm changes. We have no conclusive evidence at all that mental diseases are true hereditary characters in the strictest sense but that makes them of no less importance.

It may be said that some of the evidence often cited against the inheritance of acquired characters is really trivial. The mutilation argument, that the amputated arm, the Chinese deformed feet and the blinded eye are not inherited is like saying that because a slap in the face does not knock one down, therefore a blow with the fist cannot. Yet how often does one hear this seriously advanced?[13] Granting, as every one must, that the germplasm is very stable, that it tends to resist change, what is the evidence that influences that pour in on the body may alter germplasm so that a new generation is altered in a definite manner?

Before citing the evidence, the positive instances, certain well formulated implications of these questions become important. If the germplasm is not injured by adverse conditions, by infection, exhaustion, toxic substances, and if the weaker, less resistant and generally less desirable people are carried off by these agents, then the noblest efforts of mankind, the efforts to conquer infections, preserve the sick, ameliorate social conditions, are against the real welfare of the race, are non-eugenic in a word. And this is the position squarely taken by many very important men, who have not hesitated to say that natural selection, operating to eliminate the unfit, is impeded by modern medicine and sanitation to the deterioration of the racial stock. Haeckel and Herbert Spencer were very

[12] "Amongst the pucerons, the elodea, the potatoes and other beings capable of propagating indefinitely through non-sexual methods the differentiated cells die first everywhere, and it is only by the more or less undifferentiated elements that life continues. The fact is so general, one might say so absolute, that there is no way of disputing it."

[13] As C. Hart (74) points out, an environmental change that is to effect the germplasm must have both intensity and duration for its conditions. Mutilations certainly cannot be adduced to show that germplasm cannot be altered by the environment.

emphatic in this, and even so socially minded a man as Havelock Ellis states that it is by no means a good thing as yet for tuberculosis to be eliminated as a drastic improver of the stock although in another and later writing his point of view seems to have been altered. Throughout the writings of Pearson and the biometric school, very prominent in the works of Davenport and the American eugenists, and equally so in the writings of Martius, Baur, Bauer, Lenz, and a great many of those German writers who have concerned themselves with racial welfare appears the fear that we are headed wrong and all social amelioration is intensifying racial degeneracy. Eugenics, which has for its aim the genotype, the race, is opposed to euthenics, which has for its aim the welfare of the phenotype, the individual,—this appears very openly or covertly in much of the present day eugenical literature.

There can be no doubt that these are not necessarily identical efforts, and that what is good for the individual as we at present see it may be bad for the race. It may be good to have good hygiene, good food, good care, sufficient rest and leisure, but if this permits grossly inferior individuals to survive, and if that inferiority is hereditary, then harm to the race does result. Thus a high infant mortality may be a good thing in eliminating the unfit at the very start, and baby hygiene, prenatal care and all that sort of thing may be misguided sentimental efforts which really poisons the race. An epidemic carrying off its hundreds of thousands may be like a strong wind blowing down the sickly trees, and though horrible to contemplate may thus be of racial benefit.

I refer the reader to the admirable discussions in Shallmayer (172) and Holmes (85) for a quite comprehensive view of this part of the subject. There is no evidence that the races with highest infantile death rate are better than the others with lower death rate —Russia with its enormous infant death rate, produces no finer adult human specimens than does the Scandinavian peninsula or New Zealand with their low death rate; quite the contrary. The rural districts with their relatively low infant death rate produce certainly just as fine adults as do the huge slums of the cities with their high infant death rate. I cite H. Ellis in a thoughtful summary.

> Nor, again, must it be said that social reform destroys the beneficial results of natural selection. Here indeed we encounter a disputed point and it may be admitted that the precise data for absolute demonstration in one

direction or the other cannot yet be found. Wherever human beings breed in reckless and unrestrained profusion—as is the case under some conditions before a free and self-conscious civilization is attained—there is an immense infantile mortality.[14] It is claimed on the one hand that this is beneficial and need not be interfered with. The weak are killed off it is said and the strong survive; there is a process of natural survival of the fittest. That is true, but it is equally true that though the relatively strongest survive, their relative strength has been impaired by the very influences which have proved fatal to their weaker brethren. There is an immense infantile mortality in Russia, yet notwithstanding any resulting survival of the fittest, Russia is far more ravaged by disease than Norway where infantile mortality is low. "A high infantile mortality" as George Carpenter, a great authority on the diseases of children, remarks, "denotes a far higher infantile deterioration rate," or as another doctor puts it "the dead baby is next of kin to the diseased baby." The protection of the weak is thus in reality, as Goldscheid terms it, "the protection of the strong from degeneration."[15]

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[14] "New evidence bearing on the influence of the industrial employment of mothers on infant mortality will soon be published by the Department of Labor. The evidence, which relates to births in Baltimore, indicates that the mother's employment away from home either preceding confinement or during the infant's first year of life is detrimental to the child's health. The employment of mothers during pregnancy is associated with a high stillbirth rate, a high premature birth rate and a high mortality rate during the first month of life. The stillbirth rate among mothers who were employed in industrial pursuits during the year preceding confinement was more than twice as high as that among mothers who were not so employed. Of the live births to mothers employed, 6.2 per cent were premature, as compared with 5.7 per cent to mothers not employed. The mortality rate during the first month of life was 77.3 for each thousand among babies of mothers employed or nearly twice the rate, 39.9, among babies of mothers not employed.

That the employment of a mother, if continued until a short time prior to the confinement, is especially harmful is confirmed by this new evidence. Nearly 40 per cent of mothers employed away from home worked until within two months, and 25 per cent until within two weeks, of confinement. The employment of mothers too soon after confinement also appears to be a factor in the infant mortality rate. The mortality rate among babies included in this study whose mothers were employed away from home during the babies' first year of life was one and one-half times the rate among babies of mothers not employed." Infant Mortality and Employed Mothers. Journal American Medical Association, vol. 80, no. 13, page 948.

[15] Havelock Ellis (50).

See especially the elaborate review of the subject by Fishberg and Boas (55).

In the one great epidemic which I have witnessed at first hand there was nowhere evident a predilection of the infection for the otherwise weak or unfit. The dread influenza of 1918 and 1919, which took more lives than the great war, showed no preference for the feeble-minded, the insane, the epileptic, tubercular, the cancerous, etc. On the contrary, it took its victims right straight through the most vigorous ages of life and amongst the highest and lowest indiscriminately. It killed the young strong men training in the war camps of the United States, it struck down the young and vigorous of both sexes in factory, office, home and school. There was on the whole, if anything a lesser death rate in the schools for the feeble-minded and the insane hospitals than in the community as a whole.

An exhaustive monograph on the recent epidemic of influenza by Warren T. Vaughn (206) emphasizes some of the points in the above. It is shown that in the United States Army the cantonments on the whole had a higher percentage of infection than the tents, and on the whole overcrowdig was a very important phase in the spread of the disease. Sex played but little definite rôle, though not only was the female sex attacked in slightly greater proportion, but also the individual case appeared to have been on the aggregate somewhat more severe in that sex. All the observations agreed in finding relatively high incidence in early childhood and early adult life. A very interesting thing was found in relationship to the colored troops. Despite the fact that the death rate from pneumonia in influenza is "normally" higher in the colored than in the white race, yet the colored troops showed a decidedly lower rate than the white troops throughout the epidemic. Brewer concluded that the colored race when living under good hygienic conditions is not so susceptible to influenza as the white race under the same conditions. *In other words, what is ordinarily cited as a racial predisposition is really an environmental predisposition, something which I have tried to emphasize throughout this book.* Another interesting fact is noted. While the Irish-American stock is much more liable to tuberculosis than the Italian, nevertheless the Irish-American showed a lesser mortality and lesser incidence of influenza than the Italians did. In other words, the liability to tuberculosis by no means coincides with the liability to another respiratory disease, namely influenza. "It is sufficient to state," says the author, "that the lowest resistance

in both epidemics was in the Irish tenement districts." It is very interesting to note that the mortality is higher amongst those who come from rural homes than amongst those who come from cities, and that the new recruit is more susceptible to influenza and more apt to succumb than is the man who has been training and is accustomed to army life. "It appears that natural immunity gives way to exposure, overwork, and fatigue as was demonstrated years ago by Pasteur in his experiments on birds with anthrax. Likewise it is possible for human-beings to have their resistance lowered by exposure to unaccustomed environment." A very interesting statement is italicized by the author. "The attack rate (of influenza) showed a consistent increase as the number of rooms per person decreased." All of the foregoing which is at all definite, shows that epidemics have no special affinity for the fit or the unfit, else we must class the colored troops as more fit than the white troops; that the resistance to epidemics is a specific character, and even a definite lack of resistance to tuberculosis does not mean a lack of resistance to another epidemic; that the young and the vigorous may be selected by an epidemic and that some environmental factor such as crowding may play an important rôle.

Whatever we know about infections that is at all trustworthy is that the resistance to them is a specific quality and not at all related to the general vigor or capacity for attainment of the individual. The white man is killed by malaria and yellow fever in Africa where the black man survives[16]*—strange that eugenists have not held that up as an evidence of the superiority of the black man! It is a superiority, but in only that quality, viz., the resistance to the plasmodium of malaria and to that of yellow fever, and bears no relation to the general value of the individual.* Rats carry without great harm the germ of bubonic plague, which slaughters man by the millions. The slum immunized Jew withstands tuberculosis as the farmer Irishman does not—but the issue here is not racial worthiness but racial experience. The capacity to resist a disease is more nearly a unit quality than any of the qualities so stressed by Mendelians, it varies enormously but there is no evidence on the whole that those that perish of scarlet fever, measles, diphtheria, typhoid, tuberculosis and syphilis were inferior

[16] See discussion by Woodruff (220) "Expansion of Races."

in other than their inability to resist these diseases or otherwise unfit to survive.[17]

I return to the question I set myself—Is there trustworthy evidence to the effect that what injures the individual may injure his germplasm and thus injure the race? A priori it would seem very likely; for the germplasm is a living substance, and living in the midst of a blood and lymph stream which is greatly altered by many conditions and upon which it depends for nutrition and the maintenance of its existence. Unlike other immortals it needs the right food for life. Further it undergoes a very marked evolution before it is ready for its work of generation, and the gap between the cells lining the spermatic canals and the free floating spermatozoa is bridged over by a complex series of cells, an evolution which may, at least a priori, be interfered with at any stage.[18] This also is true of the evolution of the ovum. It is difficult for a medical man to conceive the opposite—that the germplasm is not altered or alterable—and I may say that the medical authors have quite disgusted the biologists by their "naïve" belief in blastophoria. From Morel's time, Féré, Brown-Sequard, Krafft-Ebbing, in fact all the believers in polymorphism, Kraepelin, Forel, Horsley, Adami, and a host of others have stressed this clinical point of view.

I quote the trenchant words of Adami in this regard:

> We have clear evidence that the germ cells are not after the Weismannian conception (as usually accepted) so sacro-sanct that they are unsusceptible to influences which affect the body at large. Even though their growth is restricted still they have to grow and they have to maintain existence, and in growing they must absorb and assimilate material brought to them by the blood and diffuse from the blood into the lymph. If that lymph contains soluble toxic substances the germ cells are not precluded from ab-

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[17] Again I repeat, it is not even clear that those who die of a disease show any conclusive evidence of lessened resistance to that disease. They may have had a larger dose, or a dose of a more virulent strain. Who can tell?

[18] For a very complete account of this evolution see De Lage, "L'Heredité et les grands Problèmes de la Biologie générale," pages 132 to 152, also Wilson, "The Cell," and Conklin's "Environment and Heredity." There are at least four well defined states before the more or less primitive germplasm becomes the sperm, and the same number of stages in the environment of the ovarian cells before any one of them becomes an ovum. During all these stages these cells are contained within the testicle or the ovary, and are subjected to all kinds of nutritional and toxic influences.

sorbing them as do the other cells of the body, and like these other cells by being influenced by them.

Further, the author says in his quite belligerent reaction to the biologists.

> To medical men it is a minor point whether there is inheritance of the exact defect seen in the parent who has been subjected to a given form of intoxication. The essential fact is that soluble poisons acting upon the tissues in general of the parent can act also upon and modify the germ cells, so that at some definite period such changes occur in the constitution of these germ cells by which the constitution of the offspring is permanently affected, and that their germ cells in their turn are incomplete in molecular structure.

In fact, even the theoretically fundamental question, not here to be seriously discussed, whether or not acquired characters can be inherited is not answered in the negative even by the biologists. There is a body of opinion which maintains that such changes can be inherited. One needs only to cite such names as La Marck, Darwin, Spenser, Haeckel, Naegeli, De Lage, and O. Hertwig in order to show that there is quite a respectable group in favor of such a doctrine. That living tissue can not respond directly to the environment even though that living tissue be germplasm seems on the face of it, incredible. It may be answered that incredible or not it is the fact. To this may be answered that there are facts showing that germplasm may be altered by the action of environmental influences, (1) that in certain cases these environmental influences affect only the direct descendants, (2) that in other cases with injury or alteration to one generation of ancestors several generations of descendants are injured or altered, and (3) that in a very few cases it has been shown that an injury to a parent affecting and creating a single disease character may so alter the descendants that they also have the new character. This latter, of course, is the only real inheritance of acquired characters. As I have emphasized before, we are not concerned directly with this problem. In considering the family mental disease of whatever type, whether of dementia praecox, feeble-mindedness, or epilepsy, we are concerned with the possibility that some injury to the ancestor has produced in the descendant, and may produce in several generations of descendants, a diseased condition which may grow worse and lead to race extinction. Nor is it pertinent to point out that certain types of animal experiment give negative results in this regard. It is of course

true that not all types of injury and not all conditions of life alter the hereditary germplasm. What is here maintained is that some of the profound and important conditions of life involve the germplasm as they involve the other tissues of the body, and that therefore the environment may be responsible for constitution and for inheritable constitution.

I use the term inheritable constitution in order to avoid the word hereditary, which has been defined by the biologists in such a way as to exclude the action of the environment. The general attitude taken by the followers of Weismann and biologists generally is that the types of variation are to be sharply separated from one another. For example, Baur, Fischer, and Lenz (8) describe three types of variations, (1) a paravariation coming about through the effect of the environment on the idioplasm and not transmitted, (2) mixovariation which comes about through amphimixis or the mingling of two germ cells which are not completely compatible, and (3) mutations or true variations. These mutations or true variations arise from conditions entirely within the germplasm and if favorable to the species natural selection operates to make them permanent. If not favorable to the species, the individuals so unfortunate as to carry them are destroyed.

Hart (74), who is apparently a very direct person, makes the utterly staggering remark that there can be no ground for saying that variation is spontaneously caused in germplasm without outer influences because *there is no such germplasm anywhere in the universe.* The more one considers this statement, the more powerful does it become. The fact is, there is no germplasm which is not subjected to environmental influences. *Environment is constantly bombarding germplasm.*

Baur states that constitutional germplasm-carried peculiarities may arise as follows: (1) They may be carried through the germplasm because their hereditary anlage was already present in the hereditary anlage of one or the other parents, either in a manifest or latent form. This we call heredity. (2) They may arise through incompatibility ("interferenz") of the hereditary anlage of father and mother. Thus may arise new constitutional characters not present in either parent or their ancestors. In this case we speak of amphimixis. (3) They may arise because the parental germ cells have been altered in their development through outer influence.

In this case we speak of germ alteration or germ injury, or blastophoria. This seems to me a very good statement of the case.

Most authors, especially the German writers and theoretically at least the English and American eugenists, agree that environmental influences may cause blastophoria or injury to germplasm, which may affect one generation. The American eugenists mention it as a possibility but do not give it any great importance. The London eugenists are reluctant to believe that it operates to any great extent, and in fact have carried on statistical researches as before cited, showing that alcohol on the whole has no blastophoric influence whatever. That the individual himself may be greatly altered by environment in respect to many of the important qualities is conceded. Thus Schallmayer (172), who is a Weismannian in most respects, although very comprehensive in his point of view, shows that "growth and weight may be greatly influenced by the environment."

> It is now established through innumerable measurements that these two characteristics are greatly altered by the good or bad influence of the environmental conditions, so that even with a definite hereditary anlage for these characters the developmental results may be pushed backward or forward. Especially there comes into play the nutrient and dwelling conditions, the climate and the amount of work.

He cites Von Hosslin, Martin, Ripley, McDonald, and many others. As is well known, Boas, the American anthropologist states that the head form, the basis of racial distinction, is altered by environment.

I. ALTERATION OF A DESCENDANT GENERATION BY ENVIRONMENTAL FORCES OPERATING UPON THE IMMEDIATE ANCESTORS

I do not here cite, as directly or indirectly relevant, the work of Féré, Hertwig, and many others on the individual or developing creature. As is well known Féré subjected eggs to all kinds of chemical influences and produced a wide variety of monsters. These teratological results show that the life evolution in any living egg can be altered by the environment if the environment be given access to the egg. It is of course obvious that the egg of man is not so grossly subjected to a bad environment as were the eggs Féré dealt with. Yet no one has adequately studied the progeny that survive

a human uterus infected with gonorrhea or showing that diversion of blood supply occasioned by a fibroid new growth. Where abortions and miscarriages are so common, as in syphilis, lead, gonorrhea and phosphorous poisoning, it might be worth while to study the progeny that survive the ordeal. Moreover, the poisoning of the sperm, which is the active partner in conception, though certainly not the more important[19] can injure the immediate descendants. O. Hertwig (78) and his collaborators have certainly demonstrated this on a definite experimental basis through the action of radium, etc., on the sperms of Tritonium, and it is without doubt that an inferior generation may thus arise, inferior in size, weight, mobility, and in every microscopic detail including the nuclei of the cells in medulla, heart, liver, muscle and germplasm. What this group (and many others have now established) on a scientific basis was shown as long ago as 1852 by Constantin Paul[20] in the case of the male lead worker whose wife was peculiarly liable to miscarriages and whose progeny died at birth in a very large percentage or survived in a sickly manner. T. Oliver emphasizes these facts and calls especial attention to the great deterioration of the population living in lead mining districts of Hungary.[21]

I cite in more detail pertinent researches which show that toxic substances operating upon the individual (phenotype) may injure his direct descendants. It can scarcely be claimed that the animals treated were neuropathic or psychopathic, as is the fashion when human beings and their descendants are injured by toxic agents. I am not asserting that these experiments tend to show that "acquired" characters are inherited. The point of what has been so far adduced, and of that which immediately follows is that a normal generation may give rise to an abnormal generation under unfavorable environmental conditions.[22]

[19] There is considerable literature at present on artificial parthenogenesis, or the doing away with the sperm. Classical in this matter is the work of Bataillon who literally stirred the virgin egg of the frog into fertility by a needle! Lieb used chemical means to this end. See Fritz Levy, "Uber künstleche Entwick lungserrung bei Amphibien" (122).

[20] This author also cites Lizé and Carriéré, the former in reference to mercury workers and the latter in the blastophoric results in the progeny of male guinea-pigs inoculated with the soluble products of tuberculosis.

[21] Oliver, T. (147).

[22] Cole, L. J., and Bachhuber, L. J. (28).

Alcohol has deleterious effect on germ cells of the male. In the experiments here reported lead, in the form of lead acetate, was substituted for alcohol.

A. Rabbits

Two series of experiments were run. In the first (I) a normal homozygous Dutch-marked male (♂ 20.2) and a poisoned albino male (♂ 26.8) were both bred to a number of albino females. In the other series (II) an albino male (♂ 21.4) was used as the normal or control animal, while the previous control male (Dutch ♂ 20.2) was subjected to the lead treatment.

A comparison of the two series shows: (1) The mortality of the albino young within four days after birth dropped from 47.7 per cent when the albino albino male (♂ 26.8) was poisoned to 29.2 per cent in those young which came from the normal albino male (♂ 21.4). (2) The mortality of the pigmented young in the same period rose from 18.9 per cent in Series I, when the pigmented male (♂ 20.2) was normal, to 34.2 per cent in series II, after he had received the lead treatment. (3) Coincident with the lower death-rate in the albinos in series II over those in series I, it will be noticed that there is a distinct rise in the average weight of the young at birth—from an average weight of 49.8 grams when the father was poisoned to 59 grams when the father was normal. (4) The average weight of the young of the pigmented male before he was given the lead was 54.7 grams; after the treatment the average weight of the young produced dropped to 49.1 grams. In this connection it should be mentioned that both albino males were considerably larger than the Dutch (♂ 20.2), the former varying around about 2900 grams, while the latter averaged only about 2100 grams. In spite of this his offspring averaged larger than those of the poisoned albino male.

From the foregoing it seems legitimate to conclude that the offspring produced by male rabbits which have been poisoned by the ingestion of lead acetate into the alimentary tract have a lower vitality and are distinctly smaller in average size than normal offspring of unpoisoned males.

B. Fowls

An experiment similar to that with the rabbits has been conducted with fowls. Twelve White Leghorn hens were divided into 3 lots of 4 hens each. Those in the first lot were bred only to a White Leghorn cock, which was being fed each day a certain quantity of lead acetate; those of the second lot were bred to a normal Houdan cock alone; while the hens in the third lot were bred on alternate days to the White Leghorn and to the Houdan. Thus color, comb, and toe characters could all be utilized in distinguishing the chicks of the respective cocks. Inspection of the results show that of 174 eggs obtained from hens mated to the poisoned Leghorn cock, 27 per cent were infertile, 27.5 per cent of the embryos in the 127 fertile eggs died before hatching, and of the 92 chicks hatched 13 or 14.1 per cent died before reaching the age of 3 weeks. Comparing these results with those of the normal Houdan cock, we find the percentage of the infertile eggs in the latter case is much higher, being 42.3 per cent as against 27.5 per cent. On the other hand, the percentage of dead embryos (17.2 per cent) is not much more than half as great, and the percentage of chicks dying within 3 weeks (3.7 per cent) is only about one-fourth as high as in the case of the poisoned male.

The data from mating both cocks alternately to the hens in the third lot corroborate the results above stated. In all 109 eggs were laid, of which 42.1 per cent were infertile. In 17 of the fertile eggs the embryos died before hatching. Only 10 of these could be identified, there being 9 Leghorns from the poisoned cock, and 1 crossbred of non-poisoned Houdan paternity. Of 46 chicks hatched, 31 were Leghorns, but of these 5 died within 3 weeks, while all of the crossbred survived that period. These results are interpreted as indicating that in fowls poisoning of the male parent with lead results in offspring of a distinctly lower average vitality.

II. BLASTOPHORIC EFECT OF CHRONIC LEAD POISONING (CARL V. WELLER (214))

1. In chronic lead poisoning there is a definite blastophoric effect. This can best be demonstrated upon the male germplasm in which case the blastophoria manifests itself in some instances by sterility without loss of sex activity, by a reduction of approximately 20

per cent of the average birth weight, by an increased number of deaths in the first weeks of life, and by a general retardation in development such that the offspring of a lead poisoned male remain permanently underweight.

2. The offspring of a lead poisoned female are underweight at birth and are very frequently stillborn. The number of stillbirths is out of proportion to the degree of intoxication of the mother and points either to a special susceptibility of the embryonic tissues to lead or to a blastophoric effect on female germplasm.

3. From the apparent recovery of reproductive power some time after stopping the administration of lead it seems that the deleterious effect must be suffered especially by that portion of germplasm which is undergoing maturation and not by that which is stored as undeveloped germinal epithelium.

III. ON THE INFLUENCE OF SMALL DOSES OF ALCOHOL ON THE DEVELOPMENT OF TUBERCULOSIS IN ANIMALS, WITH ESPECIAL CONSIDERATION OF THE DESCENDANTS (T. LAITINEN[23])

Alcoholized animals more quickly get tuberculosis. Agreement by Woodhead and Achard. Walter Kern says, "Alcohol exercises a harmful influence on the descendants in that (1) the general vitality is lowered, and (2) the course of a tubercular infection is unfavorably influenced" (Zeitschrift f. Hygiene, 1910, p. 455).

E. A. Homer comes to same conclusion.

Research conclusions (small number of cases):

1. Alcohol makes the animal organism more susceptible to tuberculosis (lowers the natural resistance against tuberculosis).

2. The smallest doses of alcohol 0.1 cc. per kilogram of the animal weight, given for a period of time and daily, brings about the above result.

3. The smallest doses of alcohol (above dosage) seem to hurt the descendants of the research animals.

A. Adaptation in bacteria

I cite here, though realizing that they are not completely relevant, the statements of Adami (1) in relation to the direct adaptation of bacteria. Bacteria differ from the multicellular organism in that they may be regarded as entirely germplasm (Weigert) or for that

[23] Laitinen, T., Beitrage zur path. and u. allg. path., 1911, 51, 267.

matter as entirely somatoplasm since reproduction is a matter first of growth and then of splitting into two individuals, each of which has the property of further division. Therefore when the environment changes the nature of the race of bacteria it is not acting indirectly on germplasm—it has direct access to the racial material. But the evidence Adami cites, and which Jennings finds competent, points toward the belief that variation is "primarily the result of influences operating from the environment." Adami shows that if the environment of typhoid bacilli be altered their characters are altered; change their foodstuffs, temperature, growth, etc., and they change. "Grow the diphtheria bacillus for example in milk or upon peptone broth agar and this at the same temperature, and at the end of twenty-four hours the differences are recognizable under the microscope. Nothing, in fact, is more easy to demonstrate than this capacity on the part of bacteria as a class to vary according to alterations in environment."

He cites the work of Barber in relation to the B. coli and its transition from a short stumpy bacillus to a filamentous type growing in chains; the work of Brunton, Miss Peckham, Klotz, and Twort in their work on typhoid bacilli; the work of Massini and Penfold and other observers who show that typhoid bacillus can be altered by virtue of changes in its nutrient environment, so that it will produce a race of typhoid which have gained permanently the power of fermenting iso-dulcite whereas the ordinary strain of typhoid cannot do this. He cites the classical work of Pasteur and his lieutenant Roux and Chamberlain.

> By exposing cultures of anthrax bacilli to heat a degree or two below that which will kill them, or again by the action of minute quantities of certain antiseptics added to the medium of growth, a race of anthrax bacilli are obtainable which have wholly lost the power of spore production. These races may (then) be grown for months and years, for thousands and indeed hundreds of thousands of generations upon the ordinary media of the laboratory without regaining this striking characteristic property. Were a culture of this strain given to a systematic botanist without information as to its origin he most assuredly could classify it as a distinct species. Here again is no matter of chance variation in many directions—all the individuals of the colony subjected to the particular temperature became (primarily) asporogenous.

He cites the well known changes in pathogenic organisms in the direction of increasing and decreasing their virulence, and also the

work of Vincent in showing how non-pathogenic can be converted into pathogenic bacteria.

B. Evidence to show that an environmental force operating on only one generation may continue to produce results generation after generation

1. The work of Stockard, Craig and Papanicolaou (185, 186, 187, 188). Guinea-pigs were first mated and shown to be capable of producing normal offspring before they were subjected to alcohol, and only healthy and fertile stock was employed. I am here quoting from Dr. Holmes' account of the experiment, which is very candid and complete ("Trend of the Race"):

For six days per week the guinea-pigs were subjected to the fumes of alcohol until they began to show signs of intoxication, although they were never allowed to become completely intoxicated. After this treatment was continued for some time the animals were mated. Normal males were mated with alcoholized females and vice versa; and there were also matings of alcoholized males with alcoholized females.

The immediate descendants showed a great deal of deficiency.

Out of 90 matings of normal females with alcoholized males 37 gave negative results or early abortions; 10 of the litters from the other matings were stillborn, and out of the 43 litters containing living young, about 35 lived but a few days, while the survivors, 47 in number, contained many small and defective individuals. In 33 matings between normal males and alcoholized females 7 gave negative results. Four produced only stillborn young, and of the young from the 22 living litters, 23 died soon after birth. When both parents were subjected to alcohol, out of 41 matings 20 gave no results, or early abortions. Fourteen resulted in stillborn litters, and the 17 living litters contained only 26 young of which 12 died soon after birth. Contrasted with the foregoing is the outcome of 90 matings of normal pigs giving 66 living litters with 99 surviving offspring.

One fact of much interest is that guinea-pigs from alcoholized parents produce a relatively defective progeny even though they may not have been given alcohol themselves. *Animals as far as three generations removed from the direct alcohol treatment are still differentiated as a group from the control in regard to the weight of the litters in which they are born, the tendency of the litters to result in failure, the high proportion of prenatal mortality over postnatal, and the total mortality which is one and one-half times higher than the normal.* Deformities and defects appear much more commonly in the alcoholic strains. Among these were paralysis agitans, opaque cornea, cataract and opaque lenses, small defective eyes, complete absence of one eye,

and, finally, complete absence of both eyeballs. In some cases there were deformities of the limbs, albinos, and dwarf forms with a low degree of vitality. No defects were noted in the normal line. Defects sometimes arose in strains in which the males only had been alcoholized, in some cases the treatment having been given only to the grandparents or great-grandparents of the deformed animal.

It is a noteworthy fact that when males alone are subjected to alcohol the effect on the early mortality of the offspring is often very marked, although in other respects the greatest injury is done when the females only are treated. In the latter case there is opportunity not only for the germ cells to become affected so as to produce a true hereditary change, but the embryo may be directly injured by the alcohol in the mother's blood.[24]

Deterioration in offspring as a result of intoxication of the male parent can scarcely be due to anything but a change produced in the germ cells. The fact that defects thus arising may be transmitted to further generations is indicative of the production of a true hereditary effect through a modification of the germ plasm.[25]

2. Experiments of Manfred Frankel cited by Bauer (9).

X-ray treatment on the belly of very young guinea-pigs produced not only a retarded growth in the treated animal, but in increased grade on the descendants of this animal through several generations, to the final result that by these later animals only one pregnancy comes to pass until finally the last generation remains entirely sterile.

3. C. C. Little who utilized the X-ray on other mammals has produced similar results. His paper is as yet unpublished, but in his address before the Harvard Research Club he cited experiments corroborating those of Manfred Frankel above mentioned.

[24] I think that Professor Holmes is mistaken in this. As I understand Stockard's experiments, the mating is carried on after the animal has been alcoholized, and consequently there can be no alcohol circulating in the mother's placental blood at this period. It is more likely that the mother is more important than the father in determining the general vitality and development of the young.

[25] I here cite the fact that Pearl's (153) experiments on fowls showed no such result as Stockard's on guinea pigs, but it does not essentially matter that fowls are not especially susceptible to alcohol, or does it especially matter that guinea pigs are susceptible. What matters is that some environmental influence brought to bear under experimental conditions can alter a stock for several generations. Pearl's results would be appropriate to consider if he had shown that the experiments of Stockard are not correct. All he has shown is that the germplasm of fowls is much more resistant to alcohol than that of guinea-pigs.

4. D. T. MacDougall has shown that it is possible to introduce foreign substance into the ovaries of the higher flowers in such manner that the eggs of pollen nuclei were affected as to their hereditary capacities and in a permanent way.

Many seeded ovaries with the ovules standing in an open chamber offer the best mechanical features, and only those which will recover from the traumatic effects of the necessary operations are of value to the experimenter. Satisfactory conditions were finally obtained with a scrophularia. Ovaries treated in 1911 with a solution of one part potassium iodide to 40000 of distilled water, and the seedlings grown in 1912, included two individuals unlike the parental strain. These and their progeny are unlike their progenitors in the color pattern of the flowers, the water relations of the stems, the degree of differentiation of the tissues, the shape of the stems, the form and size of the flowers, the growth correlations and venation of the leaves. *The continuance of these features in the successive generations indicates a permanent modification of the germplasm.*

5. The work of Tower. The work of Tower is exceedingly important if corroborated as bearing upon the statement that an environmental force playing upon an ancestor generation may produce apparently permanent modification of the germplasm.

Tower studied the effect of various degrees of temperature and of drying upon the potato beetle. If the pupa of the potato bugs are subjected to 35°C. of heat and drying for a certain length of time then the mature or beetle stage of the creature has a pale, abnormal coloring. Ten or 15° of temperature below the normal, that is to say cold, has the same effect, whereas a lesser degree of heat or cold if applied to the pupa brings forth in the beetle stage an increased pigmentation. What is of importance is that the next generation, the descendants of those subjected to this process, have normal pigmentation. That is, though the evolution of one form of phenotype to another (pupa to beetle) has been altered, the genotype (the race) has not been. *But if the adult beetle in which the germ-cells are ripened is exposed to heat and cold in a fashion similar to that described above as applied to the pupa, then the same changes appear in their descendants, but with this difference, that they become a hereditary character which is transmitted from generation to generation, though the adults themselves thus treated with heat or cold remain unchanged.*

Schallmayer (172) states[26] that this proves that alterations of the phenotype, or individual, do not become hereditary, while alterations

[26] Tower, cited by Schallmayer.

of the germplasm, or genotype, do become hereditary. Perhaps. The really important lesson from these simple experiments is that what happens to an individual, for surely the heat, cold or drying were applied to the individuals, may alter his germplasm and cause hereditary racial changes, a fact of vast importance. Or to phrase it differently, environment, here in the form of heat, cold, etc., may directly alter a race without the play of natural selection at all. No subtleties in terms or discussion can disguise the fact that this completely upsets the ordinary and practical implications of Weismannism.

6. I do not cite here the work of Kammerer who succeeded according to his own statement in producing through changes in the environment changes in various types of salamander in a remarkably definite manner. His work has been questioned by Bateson and therefore is at the present time of doubtful authenticity. The German writers, Hoffman and Hart, already cited, accept his results as proving that the environment can cause changes in the germplasm which persist indefinitely.

7. The work of Guyer and Smith (73). Perhaps the most spectacular and interesting experiments of all have been carried on by the Americans Guyer and Smith. Guyer and Smith injected into fowls the pulverized lens of rabbits. They then obtained a serum having a remarkably lytic or destroying effect upon the lens of rabbits. When this serum is injected in pregnant albino rabbits during the first ten to thirteen days of the pregnancy important results are obtained. Many of the foetal rabbits die. In 9 cases out of 61 of those who survived the lens was small and more or less opaque. In some cases there was a marked reduction in the size of the eyeball, and in others a complete non-development of the eye. The control rabbits injected with serum of untreated fowls showed no such modification. In other words, the blood of the fowl had obtained the property of producing a serum which passed into the placental circulation injuring the developing rabbit in a specific manner, mainly producing injuries to the lens substance and to the visual apparatus.

The most important and the pertinent fact follows:

> When this character appears in rabbits it becomes hereditary without further injury. It has been transmitted for 8 generations, and what is more important continually tends to become more serious in the successive genera-

tions and to become more common in the descendants of each generation. Further, the defective males when crossed with normal females not belonging to any of the defective families shows the following results. The first generation shows normal eyes, but the females of this generation again crossed with defective males give birth to a certain number of young with degenerated eyes.[27]

No one, so far as I know, has questioned the methods of Guyer and Smith, although I do not know of any one who has duplicated their results, but if they stand the test of time, and there seems to be no reason to doubt that they will, it does not seem possible for any one to deny that new characters may appear through the action of environmental forces, and that these new characters may be transmitted from generation to generation. Moreover, it will be noticed that in the results of Stockard and his co-workers, and of Guyer and Smith, as well as of Manfred Frankel and C. C. Little, the succeeding generations become more and more involved. That is, a dynamic process is set agoing in the germplasm, a process which shows more and more results in each generation until very marked results are shown. This is what one sees in many families with mental disease. There is anticipation and antedating, and to use an awkward word, "worsening" of the descendants. There is a striking similarity in this respect between these familial mental diseases and the experimental results here cited.

Experiments of E. Carleton MacDowell (126) (Alcoholism and the behavior of white rats. XI. The maze behavior of treated rats and their offspring). Most important of all to the psychiatrist is the work of MacDowell. His experiments lead to a conclusion that affects the whole structure of society if corroborated. After all, we are interested mainly in the conduct of people, in their capacity to learn, and in their reactions to life. The summary of his experiment follows:

An attempt was made to control or modify normal inheritance, with particular attention to mental conditions as revealed in behavior. White rats treated with maximal doses of alcohol fumes daily for twenty-eight days before and during training took more time per trial in running a circular maze than did their untreated brothers and sisters. The criteria involving perfect trials give results that tend in the same direction as those given by

[27] Guyer and Smith, "Transmission of Induced Eye Defect," Journal of Experimental Zoology, 1920, 31, page 171. Studies in Cytolysins.

the data on time, but they are less conclusive and definite. The treatment of the parents as well as the rats themselves caused no more modification in maze behavior than was found in the rats originally treated, namely, a tendency to retard learning; but this tendency with the small numbers involved is either on or below the borderline of statistical significance. A small but consistent modification of the maze behavior of the untreated offspring from treated parents was found; this appeared more clearly in the criteria from perfect trials than in the time per trial. Untreated rats from parents treated with mild doses showed no difference in their maze behavior from the controls. The following generalization is reached: Alcoholism in ancestors may modifiy the behavior of untreated descendants.

In other words, not only may the structure and the viability of an animal be altered because its ancestor has been subjected to toxic or blastophoric influences, but its conduct may be altered, its reaction to its environment may be altered. Specificially, its intelligence may be altered. This is of extreme importance in view of the statements now so common and accepted almost as a truism, that intelligence is independent of the environment.

There is therefore a respectable body of evidence for the opinion that the germplasm of an individual can be so altered and injured by environmental influences as to alter and injure his posterity for several generations. Whether that is "true heredity" or not is an academic problem which we may perhaps dismiss but with this parting shot—that heredity is the term given to cover a wide range of phenomena all focussing on the transmission of qualities from ancestors to descendants and it is only an arbitrary use of the term which excludes the effects of the environment on this process of transmission. Heredity thus may be defined so as to exclude all of the effects of the experiments above cited, but such an exclusion is merely the result of an a priori prejudice.

How can the environment in the shape of toxic substances alter germplasm? It would be easier to ask, how can it help altering it? Stockard and Papanicolaou have shown that even starving an animal produces cystic changes in its ovaries, and acute and chronic changes in testes and ovaries are found in generalized and local diseases of the human being. These alterations are easily understood by the medical man for he sees in germplasm a living substance altered as every other living tissue is by nutrition, congestion, inflammation, temperature, of local or general origin.

What seems to stand out from all of the foregoing is this: That germplasm undoubtedly is conservative and that whatever inner force it represents in the transmission of racial qualities, tends to resist deflection or alteration by the trivial matters of the environment or of existence, but it is not invulnerable and probably is not reactionary. It can be altered by disease. It can become, so to speak, diseased, and this diseased condition may persist for one or more generations and may result, as in somatic disease, in the death of the germplasm, or again as in somatic disease, may result in recovery so that the germplasm comes back to its former vitality, its former normality. A conception of germplasm such as this, which gives it credit both for staunchness in maintaining racial characters and plasticity in being modified, seems more consistent not only with the facts of life but with our very conception of life itself as something pliable and pliant.

Delage (44), Cunningham (38) and Adami (1) go much further. A theory fathered by the first of these scientists, and altered but slightly by the others, perhaps evolved independently, postulates that the germplasm is directly influenced by the condition of the various organs; that each organ of importance as it operates throws into the circulation certain substances and that these substances influence perhaps definite parts of the germplasm, and that with disease of the organs these substances, altered in amount and character, influence the germplasm and alter its potentialities. Thus there is a mechanism by which the man sick with lung tuberculosis may transmit a lowered resistance to the disease to his descendants. In a striking way this theory resembles the Abderhalden work on "abban" products. Unfortunately Abderhalden's results, and even his technique, are not at present in the best repute.

Had Weismann lived at a time like the present, which is so much occupied with internal glands, it is very doubtful that he would have made so much of germplasm and so little of somatoplasm. Today we know, that the sexual functions and development are bound up with the proper development of certain of the internal glands, that disease of thyroid, pituitary, and adrenal glands may alter the very germplasm, itself, in that ovary and testes are altered. The endocrines offer a convenient mechanism for the operation of the environment on germplasm for we have seen (page 46) that food, hygiene, etc., alter these glands. We may even project ourselves

theoretically to a very dangerous and highly shocking position—since emotion is so markedly an endocrinal matter we may even believe that it is remotely possible for continued emotional disturbance in an ancestor to injure his descendant. Thus it may come to pass that the eugenics of the future will insist that people who have in mind procreation will go into a sort of training, avoiding infection and emotional disturbance as well as alcohol, syphilis, and gonorrhea.

It is true we have not found any unanimity on the question as to whether alcohol or syphilis are markedly blastophoric, though all of clinical experience would certainly point that way. But as I have insisted before this, there is a vast field in which to search for blastophoric influences. The environment has very many ways in which it can operate upon germ plasm and no one way is theoretically excluded from adversely influencing it, just as no one toxic influence is a priori necessarily capable of injuring germplasm. That is to say, there is no basis for any conclusions to be drawn upon this matter even from a dozen types of negative experiments, for the thirteenth type may disclose an environmental factor which is capable of changing germplasm. It is essential to emphasize that the germplasm is testicle and is ovary, and that what may injure these structures may therefore alter germplasm and succeeding generations. The blastophoric influences which alter various generations may even reside in the very nature of civilization itself.

BLASTOPHORIC INFLUENCES OF THE GREAT CITY

It has been noted for a long time that residents in the city are on the whole inferior in "nervous stability" than the residents of rural districts. It may, of course, be true in any individual case that some rural district is worse than cities, and it may also be true that indivdual larger cities are better than some rural districts. It is, however, noteworthy, as Havelock Ellis (50) points out of England, that in the main the talent and genius of that country has come from the small towns and the country. I quote this author in extenso (The Task of Social Hygiene).

> And the results are not altogether unlike those which this analogy suggests. At the present time, one-third of the population of London is made up of immigrants from the country. Yet, notwithstanding this immense and constant stream of new and vigorous blood, it never suffices to raise the

urban population to the same level of physical and nervous stability which the rural population possesses. More alert, more vivacious, more intelligent, even more urbane in the finer sense, as the urban population becomes —not perhaps at first, but in the end—it inevitably loses its stamina, its reserves of vital energy. Dr. Cantlie very properly defines a Londoner as a person whose grandparents all belonged to London—and he could not find any. Dr. Harry Campbell has found a few who could claim London grandparents; they were poor specimens of humanity. Even on the intellectual side there are no great Londoners. It is well known that a number of eminent men have been born in London; but, in the course of a somewhat elaborate study of the origins of British men of genius (48), I have not been able to find that any were genuinely Londoners by descent. An urban life saps that calm and stolid strength which is necessary for all great effort and stress, physical or intellectual. The finest body of men in London, as a class, are the London police, and Charles Booth states that only 17 per cent of the London police are born in London, a smaller proportion than any other class of the London population except the army and navy. As Mr. N. C. MacNamara has pointed out, it is found that London men do not possess the necessary nervous stability and self-possession for police work. They are too excitable and nervous, lacking the equinimity, courage, and self-reliance of the rural men. Just in the same way, in Spain, the bull-fighters, a body of men admirable for their graceful strength, their modesty, courage, and skill, nearly always come from country districts, although it is in the towns that the enthusiasm for bull fighting is centered. Therefore, it would appear that until urban conditions of life are greatly improved, the more largely urban a population becomes, the more is its standard of vital and physical efficiency likely to be lowered. This became clearly visible during the South African War; it was found at Manchester (as stated by Dr. T. P. Smith and confirmed by Dr. Clayton) that among 11,000 young men who volunteered for enlistment, scarcely more than 10 per cent could pass the surgeon's examination, although the standard of physique demanded was extremely low, while Major-General Sir F. Maurice has stated that, even when all these rejections have been made, of those who actually are enlisted, at the end of two years only two effective soldiers are found for every five who enlist. It is not difficult to see a bearing of these facts on the birth rate. The civilized world is becoming a world of towns, and while the diminished birthrate of towns is certainly not mainly the result of impaired vitality, these phenomena are correlative facts of the first importance for every country which is using up its rural population and becoming a land of cities.

Further, what are the conditions of cities which entail and bring about a lowered vitality of the inhabitants? Holmes (85) says that with our unnatural indoor life, the unwholesome living of a large part of our wage-earning population, the increasing drift of people into large cities, our alcohol and our numerous diseases, it can hardly be expectd that the germplasm of the race will escape being affected in some way.

A. Unnatural indoor life of the cities

In the slums especially there is insufficient light and air. That the energy of the organism is lowered by this is without any doubt. Trudeau long ago showed that such conditions of life predisposes to tuberculosis, and that previously healthy guinea pigs became much more susceptible to tuberculosis under conditions in which light and air were excluded in sufficient amounts. Further, it has been definitely established by numerous workers, of whom I cite only A. J. Hess[28] and his co-workers Gutman, Unger and Pappenheimer, that rickets, a disease which is very common in the large cities, can be prevented experimentally by sunlight. Thus the slums not only predispose to tuberculosis and diarrheal diseases generally, but also are a potent factor in causing rickets, and rickets is without doubt back of many of the so-called spasmophilic diseases. That the dietary condition of the poor who life in the slums of big cities is markedly deficient in vitamines, in green vegetables, and in those subtle constituents which come from the soil, is another factor making towards race degeneracy. There is evidence that any change in environmental conditions may even make animals sterile as is well known by the keepers of menageries. There is work being done at present which shows that it takes but a seemingly moderate impoverishment of food to bring this about.

Sunlight gives an increase in inorganic phosphorus in the blood. Rickets in rats may be prevented by sunlight despite the use of an otherwise ricketic diet. Those who wish to read an impassioned yet on the whole logical arraignment of the slums of big cities and bad urban hygiene in general as a factor in race degeneracy are referred to C. W. Sallebey's book.

B. Civilization is syphilization

This is a trite saying amongst medical men, and refers to the increase of syphilis under conditions of urbanized commercial life. There is no doubt whatever that syphilis is much more prevalent in the big cities proportionately than in the small cities. All statistics on this matter are in agreement and all writers here (as almost

[28] "Experimental Rickets," A. J. Hess, L. J. Unger, A. W. Pappenheimer, and P. Gutman, Proceedings of Society for Experimental Biology and Medicine, 1921 and 1922, pages 8 and 32.

nowhere else) coincide in their conclusions. Commerce, transportation, the big cities, are in league with prostitution to bring about syphilis. Blaschko and Fischer[29] cite the great increase in venereal disease that has taken place in the cities. They find that industrialization, and especially of women increases it. Even more than soldiers and sailors do waitresses, saleswomen and salesmen have these diseases. Wherever greater freedom exists, wherever there is less regard for conventional morality there is a greater increase of syphilis. If syphilis is a blastophoric influence injuring the race, then no matter whether or not conventional morality is justified on ethical grounds, it is justified on hygienic and eugenical grounds. Free love fosters free syphilis.

There is not much doubt but what has been said above of syphilis is also true of alcohol. Alcoholism has its greatest incidence in the cities, its lesser incidence in the rural districts. Whether or not alcohol is a blastophoric influence it is unquestionably one of the phenomena of the great city, though it has its roots in the rural districts and existed prior to the rise of the big cities.

It may be that the sum total of influences of the great city, its stimulation, its excitement, its greater stress and strain, its lesser restfulness, lowers the vitality of the organism, and lowers the total energy in more subtle ways and in ways which may have their effect upon the germplasm. This, of course, is entirely a hypothesis. The smaller size of families in big cities may be mostly due to voluntary race limitation, and yet it is possible that man like other animals becomes less fertile, less vigorous genetically under the conditions of town life. Bonhote (19) has written an interesting book on vigor and heredity in which he relates the hereditary capacities to changes in the general vigor of the animal. Undoubtedly he is mistaken in ascribing vigor to the rate of metabolism except in some rough way, for under very much increased rate of metabolism vigor declines, as in hyperthyroidism. Yet there is a good deal of evidence to show that immature parents have a rather larger number of defective descendants than mature parents, and this is undoubtedly due to the general vigor of the organism and the state of development of the germplasm.

A very eloquent, rather passionate critic of the Herodian school of Eugenics, as he calls those who believe in the operation of disease,

[29] Archives for Rassen und Gesellschafts-hygiene, 1915, 11, 408.

etc., as a means of bettering the race is Dr. C. W. Saleeby (168). In a book (the Eugenic Prospect) he emphasizes blastophoria as few have done, and finds it in many things which the ordinary eugenist passes over with a sniff of scorn. I quote his words in relation to the cities.

> Cities of our present sort are racially fatal. Their darkness, due to smoke and its consequences, we will later discuss; but they also tend to involve the breaking-up of the home, the decadence of breast-feeding, the degradation of adolescence, the rapid spread of knowledge of contraception, which latter I do not regard as inherently evil, but which is more prone to ruinous abuse than any other form of knowledge I can name. Our cities have been carrying on by the immigration and destruction of healthy young rural life so long that the rural resources are well-nigh drained—we are probably 85 per cent urban now—and unless we can reconstruct our urban ways, as, thank Heaven, one may see them being reconstructed all over North America, we are doomed.

There are many who believe that the very nature of civilized existence itself, thought, the elaboration of stimuli, the increased excitement, the stress and strain of consciousness help bring about mental instability. Conklin states that the great prevalence of nervous disorders in the intelligent class of the present day indicates that the nervous system has already developed a point where it is going out of balance with the other vital functions. Bleuler too sees in the conditions of civilization a predisposition to mental disease and race breakdown. Bron believes that the worst conditions which surround the human race are not alcohol, syphilis, etc., but are those industrial and social conditions of the large cities which injure the nervous system, and lower digestion and nutrition. He thinks that the organism is poorly adapted for intellectual overwork.

Whether the large city is or is not fundamentally bad,[30] there is no question that one phase of the large city, the slums, is unqualifiedly bad. Undoubtedly there will come a time generations hence when men looking back on the century in which we live will say,

> There was a time and a generation which knew how to fly in the air and dive under the waters, knew how to shatter nature's obstacles by explosives, was acquainted with the infinitely large and the infinitely small, and was foreseeing a future which was to release sources of energy of incalculable

[30] For a moderately toned review of this subject see S. J. Holmes, Sci. Monthly, 1919, 8, p. 16–31.

power; in that time there was mechanical skill, wealth, beauty and a keen intellectual life—and yet the men of that time tolerated the slums and permitted a large part of the race to fester and stew in great sunless brick and mortar hovels.

I am fully convinced, though of course I cannot at all prove, that the race suffers with every increase of urbanization so long as it means an increase in slums. It is possible, no doubt, to conceive of large cities spread over great territories made up of magnificent buildings with the access to sunlight, access to green grass, and access to fresh food supply, with no slums, with little alcohol, syphilis, free from stress and strain. When such a large city comes a good deal of blastophoria will disappear.[31]

BLASTOPHORIA AS A WORKING HYPOTHESIS

The hereditary transmission of mental disease is a problem of psychiatry, to be attacked in the main by clinical-experimental methods. Some day when we have data sufficiently accurate and representing the fruits of a more intensive study of individual cases it will be possible to call on the statisticians and the biometricians to help formulate laws, but that day is far off.

As a working hypothesis it seems to me more logical to search the environment for the causes of family mental disease than to fall back on "pure heredity." In the first place the environment offers

[31] It is pertinent at this place to call attention to the work of Henry A. Cotton (32, 33, 34), Director of the New Jersey State Hospital for the Insane, and his colleagues. Cotton's work rests primarily upon the belief that inheritance or heredity plays but little rôle in mental diseases; that focal disease of one type or another, whether in nose, mouth, gastrointestinal tract, genitourinary tract; in fact, anywhere in the body, may bring about the crumpling of personality that we see in dementia praecox or the alteration in mood that we note in manic-depressive psychosis. In other words, he brings down the mental diseases to a definite organic basis, not in the nervous system but in the bodily organs and having its affects upon mind and personality in a way perfectly understandable in the light of modern science. He claims the most extraordinary results from surgical treatment, from the extirpation of the foci of disease. His work has been vigorously attacked, especially by Kopeloff and Cheney, who deny the reality of certain of his findings. So far as I know, no one has corroborated him. Nevertheless, extreme as his statements may be, they develop a line of investigation which deserved commendation and at least an effort should be made to check up his results and to duplicate them.

a more hopeful prognosis, for it will be easier for mankind to alter the environment than to induce the geniuses, the queer, the nervous, the criminal, the alcoholic, the migrainous, the gouty, etc., to refrain from sexual life and parenthood. If we too readily assume heredity of an inevitable kind as cause, the result is a paralysis of investigation, for any fundamentally fatalistic doctrine inhibits research and study. But if we say that the environment, in some of its forms, as toxin, infection and lowerer of vitality, acts in a blastophoric way we are stirred to research and study, and results must follow.

A program of research in the inheritance of mental disease will then be clinical and pathological. It may tentatively be carried on as follows:

1. An intensive physical and mental study of relatives, especially of parents, and this to compare then with an intensive study of the patients. Field workers to be used, but their results are only to be a beginning of investigation, and they are to operate mainly to bring the parents and relatives in for study. Height, weight, shape of head and body, Wassermann reactions, endocrinal studies—these and others to be made on parent and child. This investigation to be made especially in those cases where apparently normal parents bring forth children with mental diseases.

2. An intensive study of the conditions surrounding the birth of the patients. Whether or not either parent suffered in any unusual manner, or there was any decided fluctuation in strength and energy; whether there was some parental infection prior to the birth of the child or during pregnancy—a search for blastophoric history.

3. If possible studies of seminal fluid should be made of parent and child. This of course would be difficult but not at all impossible.

4. Postmortem studies should pay more attention than has been the rule in insane hospitals to the generative organs and also to the endocrinal organs. Most studies in the insane hospitals have laid too much stress on brain and not enough on testes and ovary.

5. Antemortem studies may well emulate the work of Henry Cotton and his colleagues. Even if their work be wrong in every result it is more essential than a million studies carried on by field-workers operating to substantiate pre-conceived opinions.

In other words, intensive and comparative clinical studies, such as have long been carried out in an individual way by William W.

Graves (70, 71, 72) of St. Louis and whom I gladly acknowledge as the source of whatever zeal I have in family studies, offer a reasonable hope for light on this dark problem.

I call attention at this point to the work of Graves as highly interesting and highly significant. In 1910 he described two types of scapulae observed in human beings. The first is the convex type, so named because its vertebral border is convex, and having in addition a great many other characters of a definite type. In marked contrast to this, he described a concave scapula, also known as the scaphoid scapula, the most striking characteristic of which is a concave vertebral border, although, as with the convex type, there are many other characters of importance. Intermediate between these two types is the straight type of scapula which belongs fundamentally to the concave type because its characters resemble the concave type more nearly than they do the characters of the convex scapula.

Graves points out that these types of scapulae are found at all stages of life and are found in essential purity in the fetus. A striking statistical observation as to the incidence of these three types of scapulae at the various stages of life was discovered by Graves and confirmed by many writers. This fact is as follows: The concave type of scapula and the straight type of scapula predominate in childhood but grow progressively less frequent as time goes on, in old age the convex type of scapula is more common than the other types. This observation brings up at once the question whether or not the type of scapula changes in the same individual during life as the result of activity or muscle stress and pull. Graves answers that it does not change; first, because these types are found as characteristically in the fetus as in old age, and second because he has records of observations on many individuals carried on since 1910 in which the type of scapula has not changed at all in the transition from childhood to adult life. This fact, that the concave and straight type are more common in early life and grow progressively scarcer as time goes on, would indicate that people with these types of scapulae are relatively more vulnerable than the people with the convex type of scapula, that they are carried off by disease processes in greater proportion. If this is confirmed by the test of time then Graves has discovered a fact of great importance, an outward sign which indicates the constitutional viability of individuals. In re-

lationship to the heredity of these scapulae, Graves points out that the inheritance of the type of scapula is a blend between the types of scapulae of the parents. If the scapulae of the parents are alike, the children tend, under normal circumstances, to have the same type of scapulae as the parents. On the other hand, if the type of scapulae of the parents is unlike, then the scapulae of the children tend to be intermediate in type. Graves, however, has made the interesting observation, that if some blastophoric factor is at work, the type of scapula seems to be altered towards the scaphoid or concave type. It has erroneously been assumed in the literature, that Graves considers the scaphoid or concave type of scapula, a stigma of congenital syphilis. This is by no means true, either of congenital syphilis, or of the view point of Graves.

Such a research demands an institution, equipped with men, money, and the spirit of science, utilizing every known technique, studying the environment and the germplasm not as independent factors but as interpenetrating each other in a complex way. Such an institution would contribute to the welfare of society more than can be estimated in dollars and cents, for its prime object would be to study the forces that operate to dethrone the mind of man and its results would contribute to the dignity and value of existence. Though that institution may be far off, or just around the corner, we who are concerned with this problem of psychiatry may well labor in laboratory and clinic, in experiment and postmortem examination, to discover the ways in which disease and bad environmental conditions of all kinds operate to injure germplasm.

Our work should, of course, not end with any programme interested only with the anomalies transmitted from one generation to another. The whole problem of constitution needs to be studied with a mind freed from the prejudice that constitutional anomalies are necessarily inherent. Durken and others have shown that a local injury to the developing embryo has widespread general developmental effects and that the earlier such injury is inflicted the more the whole constitution is changed. Studies of constitution on something of the plan carried out by Bauer and Kretschmer (115) in Germany and Goldthwait and Bryant in America but extended, are even prior in importance to studies on heredity.

In other words, a study of those environmental forces which alter character and the general trends of the physical and psychical life

of individuals must be linked up to a study of those environmental forces which alter these sets of qualities in a family group or the race. That long and arduous studies await us before we can even prepare to understand the problem of family mental disease needs no argument, but that is only another reason why they must be made. And especially they must be made before we leap into legislatures with demands that this or that measure be carried out, before we call for the wholesale sterilization of the feeble-minded, the insane, the epileptic, and the criminal as blithely as if we knew all about the inheritance of mental disease when indeed we know remarkably little. We have a right, I think, to pass laws that no one shall conceal the fact of mental disease when entering upon marriage and that the concealment of such disease shall be a cause for annulment, whether or not the individual was insane at the time of marriage. We have a right to ask for the sterilization of those types of feeble-mindedness which we know to run in families. Wherever mental disease exists in a family group for more than one generation, it would be wise for society to sterilize those of the second generation who go to institutions. I do not believe that we can ask much more than that in the present state of our knowledge and we cannot afford to be unduly dogmatic. The common sense and stolidity of legislatures, what seems to be stupidity, is often enough justified by the dogmatic attitudes of science, a dogmatism hard to understand by one who knows the history of the theories of science.

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